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ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

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LXXXII

THE DEVELOPMENTAL ANATOMY OF THE HUMAN STAPES*†‡

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AND
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CHICAGO

In the course of its phylogenetic history, the stapes has passed through a remarkable series of transforming stages; these, to an extent, are repeated in the individual developmental history of this ossiculum in man.

The predecessor of the human stapes was part of the skeletal support for the respiratory mechanism in aquatic and amphibian creatures; in mammals it has become auxiliary to the organ of hearing, being made over to serve as the terminal element in a three-link ossicular chain whose function it is to convert air waves into liquid waves. The stapes, therefore, in each individual must develop initially as part of the branchial arch system, but must establish new

*From the Department of Anatomy and the Department of Otolaryngology, Northwestern University Medical School; contribution No. 381 from the former.

†Read before the Chicago Laryngological and Otological Society, April 6, 1942.

‡Investigation conducted under the auspices of the Central Bureau of Research of the American Otological Society.

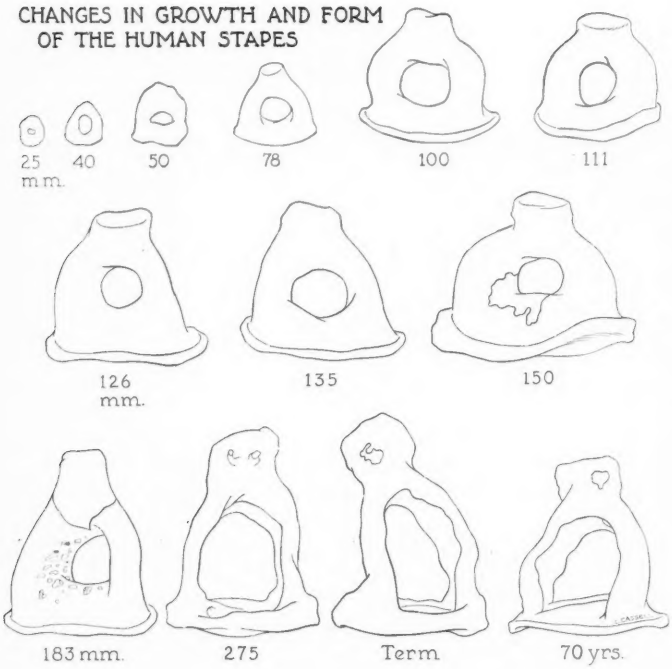

Plate I.—Drawings of reconstructions of the human stapes. Measurements, crown-rump length of embryos. x1/6 (approx.).

and anatomically important relationships at one extremity with another ossicle, at the other with a window in the capsular house for the end-organs of hearing; the stapes must be well-supported by articular ligaments and muscles, yet free to take vibratile excursions; it must lose some of its bulk in order to reduce inertia, but without reduction in physical strength; the stapes must develop rapidly to be ready for inclusion in an orifice whose dimensions are fixed at the middle of fetal life, but must cease growing abruptly as soon as its base fits the fenestral opening; it must adapt itself to an existence within a chamber invaded by pharyngeal mucous membrane, its whole vascular supply being altered in pattern to fit a submucosal existence.

The characteristic adult form of the stapes is explicable in the light of its developmental history. The major features in stapedial morphogenesis will be the subject of this paper.

The present account is based upon a study of the extensive series in the collections at the University of Wisconsin, the Carnegie Laboratory and the Northwestern University Medical School. The Wisconsin series were made available through the courtesy of Dr. Theodore H. Bast, those of the Carnegie collection through the kindness of Dr. George W. Corner. In all, 140 series were studied (30 Carnegie, 50 Wisconsin, 60 Northwestern).

Reconstructions were prepared from series which represented crucial stages in stapedial growth and differentiation (Plates I to IV); they were supplemented by photomicrographs illustrating two important phases of morphogenesis, namely, conversion of the ring-shaped primordial "stapes" into a stirrup-shaped cartilage of conventional structure, and of the latter into a highly specialized ossicle (Plates V and VI).*

The human stapes makes its appearance as a concentration of mesenchymal cells in the 7-mm. (4-week) embryo.† The stapedial blastema arises from the cranial extremity of the second branchial bar (hyoid or Reichert's bar). It is wholly independent of the primordial capsule, which is a meager cellular condensation around the otocyst. At 22 mm. (7½ weeks) there is still an uninterrupted mesenchymal connection between the stapes and the hyoid bar (Plate V, Fig. 8 at asterisk). This hyostapedial connection persists,

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*The figures in Plates I to III are drawings of wax-plate reconstructions prepared at a magnification of 125 diameters from series of sections in the collections at the University of Wisconsin and at Northwestern University Medical School. These three plates are used through the courtesy of the Quarterly Bulletin of Northwestern University Medical School. The drawings were prepared at considerable reduction, and further reduction occurred in reproduction. The diagrammatic drawings in Plate IV do not represent actual specimens or reconstructions. The photomicrographs in Plates V and VI were reproduced without reduction.

†Details of macroscopic form and microscopic structure, covering some of the stages described herein have been presented in earlier articles in the *Archives of Otolaryngology* (28: 1938; 29: 1939) and in the *Quarterly Bulletin of Northwestern University Medical School* (14: 1940; 15: 1941); additional material concerning this stage and others is discussed in two articles in press, *Archives of Otolaryngology*. The present article, being of the nature of a summary of this investigation, will not refer again to the individual publications just noted.

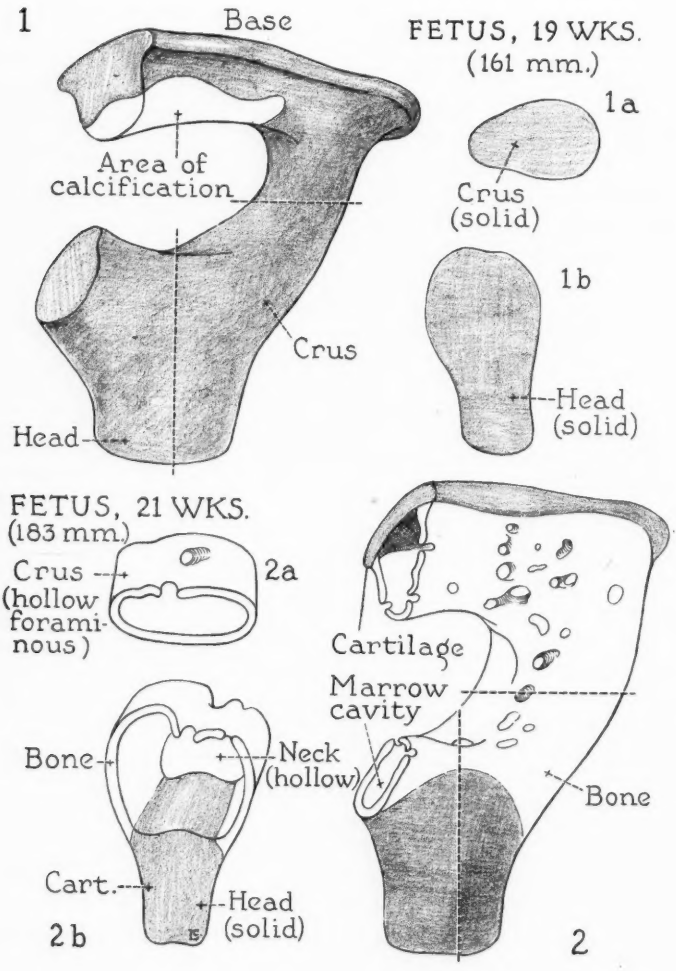

Plate II.—Semidiagrammatic drawings, based upon reconstructions, depicting major features in the development of the stapes.

as mesenchymal tissue, through the 30-mm. stage; it later becomes not a cartilaginous element, but the tendon of the stapedius muscle (Plate VI, Fig. 10). At the 50-mm. (11-week) stage the stapes has lost its connection with the branchial skeleton; but articular association has already been established laterally with the incus, and medially, by impingement, with the layer of tissue occupying the site of the vestibular (oval) window. The tissue closing the "window" gradually contributes cells to the tympanic part of the stapes; peripherally this tissue becomes the annular ligament. Thus beginning as a branchial element, the stapes becomes a capsular associate. At this stage the mucous membrane has advanced to the stapes, but has not yet invested it. In the 78-mm. (13-week) fetus the definitive portions of the stapes are clearly recognizable; the short cylindrical head and the flattened base are set off from the diverging crura (Plate I). These morphological features are well demonstrated in the 100-mm. (15-week) fetus (Plate I). The stapedius muscle has appeared, passing from the stapedial head to the capsular wall. The stapes is still entirely cartilaginous.

In the embryo of 25 mm. (8 weeks) the stapes is a complete precartilaginous ring, its shape thus differing from that of the chondral predecessor of any long bone (Plate I). In the embryos of 40 mm. (9½ weeks) and 50 mm. (11 weeks) the primordial annular form is altered, the medial aspect becomes flattened, the lateral aspect cylindrical, and the portion of the ring between these extremities is somewhat bowed; in the 100-mm. (15-week) and 111-mm. (16-week) stages, the above characteristics have simply progressed to clearer definition (Plate I). The stapes of the 126-mm (17-week) fetus is, except for size, like the preceding; there is a progressive increase in length and width, and in thickness of the several parts. Individual variations occur even among these early stages: the stapes of the 111-mm. fetus is not as far advanced as that of the 100-mm. fetus in the development of characteristic stapedial features; similarly, the stapes of the 126-mm. stage presents a massive base, exceeded in length only by the 150-mm. (18-week), and in width by the 183-mm. (21-week) specimens. These differences in size of the cartilaginous stapes are entirely within the normal range of anatomic variations regularly encountered in osseous specimens from adults between twenty-five and seventy-five years—some excised stapes being one and one-half times as large as others.

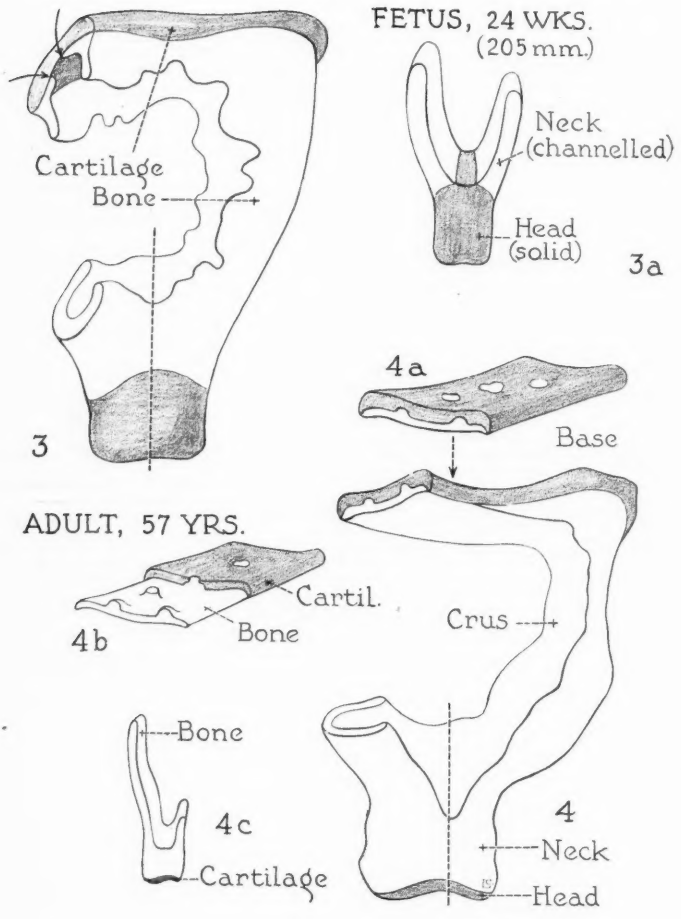

Plate III.—Semidiagrammatic drawings, based upon reconstructions, depicting major features in the development of the stapes.

In the stapes of the 150-mm. fetus and of the 161-mm. (19-week) fetus osteogenesis has just begun (Plate II, Fig. 1; Plate I).* The solitary ossification center is irregular in outline and basal in position. The crura (Plate II, Fig. 1*a*) and head (Fig. 1*b*) are still solid cartilage. Thus, while in long bones ossification is initiated at or near the middle of the shaft to form a collar of primitive bone, in the stapes it is begun in the flattened, basal part of what was originally a ring of cartilage; therefrom it spreads along the crura to the head. In the stapes no secondary centers of ossification occur; stapedial development, therefore, resembles that obtaining within the diaphyseal portion of a long bone (Plate IV, Fig. 6).

As soon as these "diaphyseal" features are established, the ensuing formative stages become so specialized that they serve to mark the stapes as a unique skeletal element. With the formation of a periosteal shell around an excavated cartilaginous core, the mechanism is set in operation for the conversion of a solid chondral member, of stapedial shape, into a hollowed osseous structure of similar size and outline—a modified ring without epiphyseal interruption. In the 183-mm. fetus progressive invasion has resulted in the destruction of cartilage with the exception of that on the vestibular aspect of the base and the incudal surface of the head, laminar and cylindrical, respectively (Plate II, Figs. 2 and 2*b*). The crura, base and neck of the stapes now constitute parts of a tubular bone the space of whose hollowed interior is continuous circularly (Figs. 2, 2*a* and 2*b*); the osseous wall is continuous around the circumference of the obturator foramen. At the same time a further step in erosion is initiated which is peculiar to stapedial development: the

*In Plate II, Figs. 1*a* and 1*b* represent sections through the solid cartilage, Fig. 1*a* through the crus transversely and Fig. 1*b* through the head coronally; the levels of cut are indicated by dotted lines in Fig. 1. Figs. 2*a* and 2*b* are comparable sections through the crural and capital portions of the hollowed osseous stapes (Fig. 2).

In Plate III the coronal sections (Figs. 3*a* and 4*c*) through the channeled stapes of an advanced fetus (Fig. 3) and of an adult (Fig. 4) are to be compared with the hollowed (Fig. 2*b*) and solid (Fig. 1*b*) stages in stapedial morphogenesis. The sections (Figs. 4*a* and 4*b*) through the base of the adult ossicle demonstrate the bilaminar character of the flattened vestibular aspect of the ossicle (Fig. 4); in Fig. 4*b* the cartilaginous lamella has been partially removed to show the osseous lamella.

In Plate IV the treatment is diagrammatic; the pictured change in length of the typical bone (Figs. 5, 6 and 7) is intended to suggest increment, but does not record correct proportional growth for the several stages in differentiation. It should also be pointed out that the representations of the typical bone (Figs. 5, 6 and 7) do not portray developmental stages identical with those of the stapes (Figs. 5*a*, 6*b* and 7*b*). The drawings of the stapes are based upon the 25-mm (enlarged) 183-mm. and 275-mm. (to scale) stages.

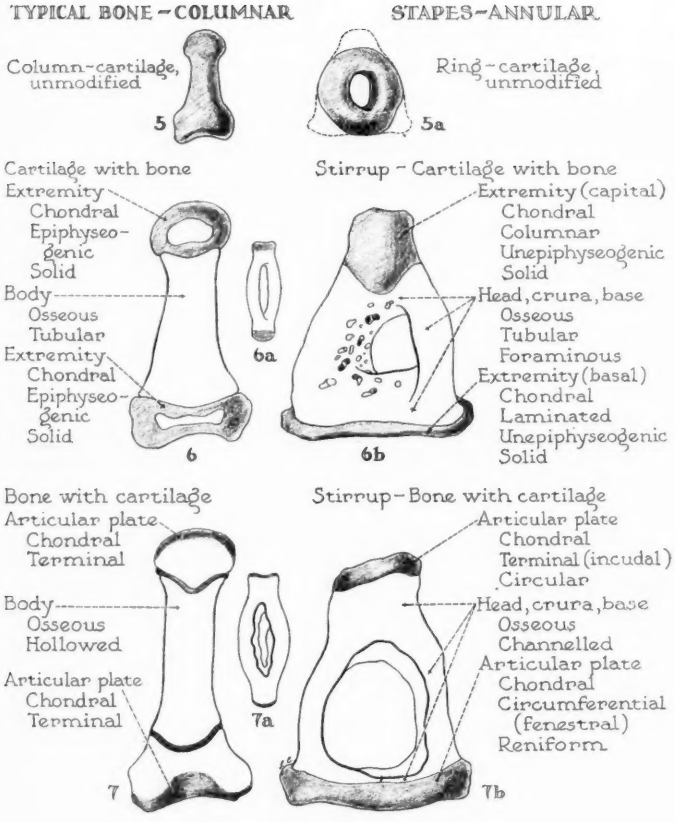

Plate IV.—Diagrammatic drawings, illustrating in comparative sets, the development of a typical bone, of a hypothetical long bone, and of the stapes.

surfaces of the entire stapes which face the obturator foramen—that is, the internal aspect of the original ring—are destroyed by osteoclasts, leaving a foraminous inner wall (Fig. 2). The stapes may now be likened to a hypothetical long bone which is incompletely bifid and diaphyseal in character, since it lacks epiphyses (Plate IV, Fig. 6*a*).

It will be remembered that the precartilaginous ring of the early embryo (Plate V, Fig. 8) undergoes rapid conversion into a stirrup-shaped cartilaginous "ossicle"; it is still entirely cartilaginous in the 126-mm. fetus (Fig. 9) and so remains until the 150-mm. stage, at which time bone makes its initial appearance in the basal portion of the stapes. Spreading therefrom it produces an osseous shell, the latter enclosing spicules of endochondral bone, primitive marrow and residual cartilage (at 190 mm. or 22½ weeks; Fig. 10). At this stage the stapes resembles, histologically, the adjacent portions of the otic capsule. However, the succeeding steps in structural modification of the stapes are strikingly divergent from those which characterize the maturation of a regular long bone. The effects of these novel steps in stapedial morphogenesis are evidenced in the fetus of 205 mm. (24 weeks). The internal periosteal wall of the crura, base and head are entirely removed, together with all endochondral bone and associated marrow (Fig. 11); the base becomes a flattened plate, the crura guttered columns, and the neck an excavated cylinder (Plate III, Figs. 3 and 3*a*). The external persisting wall of each crus is thin; the base likewise is markedly thinned and flattened. Cartilage persists on the two extremities, laterally forming an articular surface for the incus, medially being articular on the circumference of the base. On the inner surface of each persisting cartilaginous lamella bone spreads across the gap as it does in closing the extremities of a diaphysis, with the result that an osseous layer is added to a thinned chondral layer (Fig. 3 at arrows, and Fig. 3*a*). The persistent cartilage plates—capital and basal—thus serve as foundations to which bone is applied; they are not areas of epiphyseal growth. These alterations are accomplished without over-all increase in size; once bone formation has produced a complete ring—i.e., an osseous annulet modified to possess head, crura and base—subsequent changes are in the nature of reshaping, not of enlargement. Unlike a long bone, stapedial enlargement ceases as soon as the stapes is formed in osseous tissue. From a modified osseous ring, the ossicle is thus converted into a truly stapedial element whose open portion is continuous along the obturator circumference (Plate III, Fig. 3); each crus

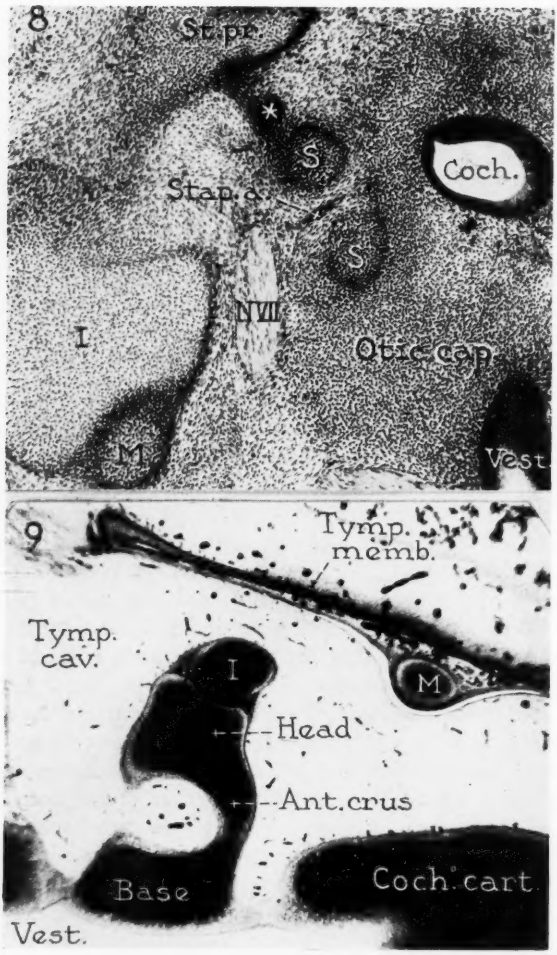

Plate V.—Photomicrographs of the stapes and related structures in human embryos. Fig. 8.—22 mm. (Carnegie No. 4960), x85. Fig. 9.—126 mm. (Wisconsin No. 11), x23. *Ant. crus.* Anterior crus of the stapes. *C.* Cartilage (hyaline). *Coch.* Membranous cochlea. *Coch. cart.* Cochlear cartilage. *E. B.* Endochondral bone. *I.* Incus. *M.* Malleus. *N. VII.* Facial nerve. *Otic cap.* Otic capsule. *P. b.* Periosteal bone. S. Stapes. *Sc. V.* Scala vestibuli. *St. lig.* Stapedial ligament. *St. pr.* Styloid process. *Stap. a.* Stapedial artery. *T.o.f.a.f.* Tympanic orifice of fissula ante fenestram. *Tymp. cav.* Tympanic cavity. *Tymp. memb.* Tympanic membrane. *Vest.* Vestibule. Asterisk indicates hyostapedial ligament.

corresponds to a long bone halved longitudinally (Plate IV, Figs. 7, 7*a* and 7*b*).

In its major features the stapes is now an adult ossicle. Once definitive form is established in late fetal life, alteration proceeds at a retarded pace. Cartilage slowly gives way to bone; while in the fetus at term, in the infant and in the child, cartilage covers the entire vestibular aspect, in adult stages osseous tissue presses through the cartilaginous basal lamella, to appear on its medial surface as islets of bone (Plate III, Figs. 4*a* and 4*b*). In young adults these patches are small and isolated; in old adults some of these coalesce, so that bone occupies approximately half the surface. Cartilage remains at the capital extremity only as an articular layer (Figs. 4 and 4*c*). On the nonarticular portion of the head and on the adjacent area of the neck of the stapes the bone often becomes very thin, sometimes perforate. Such erosion may be observed not only in the adult and in the term fetus, but also in prenatal stages (Plate I: adult; fetus at term; 275-mm. or 31-week fetus). Assumedly, weight must be reduced wherever loss of substance does not bring structural weakness.

Lacking the customary mechanism of lengthening and expanding, the stapes has attained adult dimensions, when the remainder of the body is still fetal; in the middle of intrauterine life, when the typical long bone is still destined to grow for two decades, the stapes is approximately as large as it will ever be. Already accommodated to a fenestral orifice in the otic capsule, articular relations are well established at an early age. Such slight transformations as do occur during the second half of fetal life and during postnatal years are the result of exceedingly slow histologic rearrangements.

Authors regularly include the stapes in the category of bones which arise through the gradual replacement of cartilaginous models by osseous material—a category which includes all the bones of the extremities, vertebral column and thorax, and the greater part of the skull. But the stapes, as has been shown, departs very early from the conventional developmental plan: its primordial form is that of a ring (Plate IV, Fig. 5*a*), not that of a column (Fig. 5); its full size is attained, by rapid growth, in the midterm fetus, while long bones increase many times in length and diameter thereafter; it has approached its adult dimensions as soon as its rapidly growing cartilage model begins to undergo ossification; and because its periosteum becomes relatively inactive, the stapes cannot broaden. Departing still further from the developmental plan which is ad-

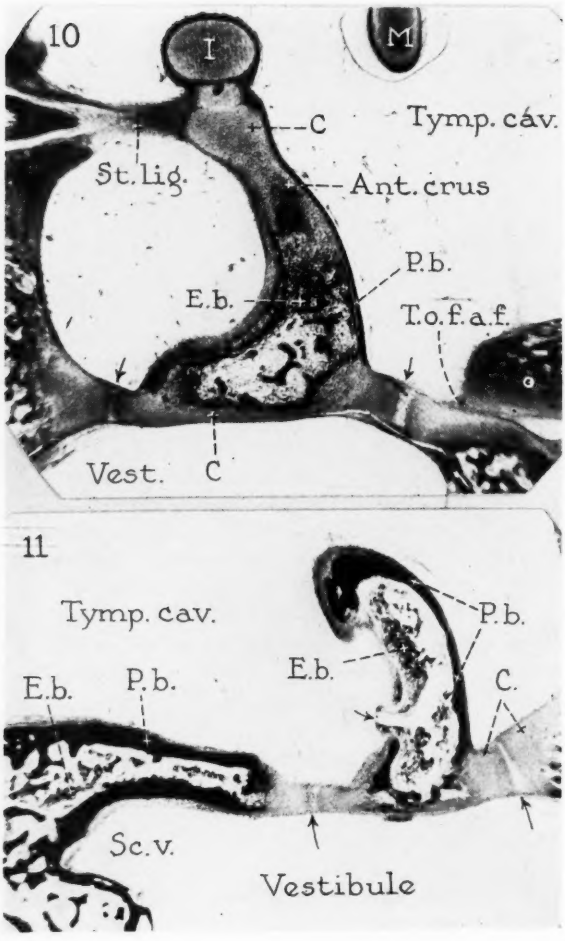

Plate VI.—Photomicrographs of stapes and related structures in human embryos. Fig. 10.—190 mm. (Wisconsin No. 29a) x23. Fig. 11.—205 mm. (Wisconsin No. 129), x23. Vertical arrows indicate margins of vestibular window; horizontal arrow, blood vessels to marrow tissue.

hered to by other skeletal units, the longitudinal half of each crus is absorbed; while still a fetal structure, marrow and endochondral bone are completely removed.

CONCLUSIONS AND SUMMARY

The stapes makes its initial appearance in the embryo of 7 mm. (age, 4 weeks). The first lunar month of its existence is occupied in a gradual dimensional increase, and in a concurrent process of conversion of a mass of mesenchyme into a distinctly annular element with a clear-cut central foramen. Not until the cartilaginous mold has been established at 30 mm. does a period of rapid increment occur. During the succeeding twelve weeks the stapes approaches the limit of chondral growth, and subsequent ossification results in crystallization of shape and size.

The characteristic stapedial form is foreshadowed in the cartilaginous "ossicle" of the 40 mm. fetus, and is definitely established in that of the 78 mm. stage. By 150 mm., and with considerable growth during the interval, ossification is initiated on the obturator surface of the base; spreading to the crura and involving the obturator aspect of the head, the ossifying process, in the 183-mm. stage, replaces all cartilage except that on the vestibular surface of the base and the tympanic surface of the head; bone surrounds the obturator foramen, forming an osseous ring uninterrupted by epiphyseal cartilage. Thus established in bone by the middle of intrauterine life the stapes thereupon ceases to grow perceptibly. No other bone in the human body is an "adult" bone—so far as dimensions are concerned—when the remainder of the body is still fetal. The stapes develops from cartilage into bone from a single center of ossification. It is at first ring-shaped, suggesting the shaft of any long bone—femur, humerus, or tibia—bent in annular form. But unlike any of these, the inner surface of the modified ring becomes eroded, so that the obturator aspect is rendered foraminous, then guttered to resemble a diminutive diaphysis which has been split lengthwise, deprived of its cancellous tissue and marrow and invested by mucous membrane. Bone grows across the cartilage plate at the base and head, whereupon the extremities become bilaminar. From the inner, osseous layer bone slowly encroaches upon the cartilage of the outer layer, with the result that osseous tissue presses through the capital and basal chondral plates to appear, respectively, on the incudal and vestibular surfaces. Lacking epiphyses, the stapes cannot lengthen; lacking productive periosteum, it does not widen. Its size is fixed by the fifth fetal month, the

adult form established at the end of the seventh; growth of the stapes thus ceases concurrently with that of the otic capsule, that is, when the fetus has passed through but half of its intrauterine existence.

The stapes, then, is unique in its developmental history; it is the only annulet in the human body, and the most exquisitely fashioned bone in the entire supportive system. Beginning as an obscure cog in the respiratory machine, it becomes an almost indispensable part of a sensory apparatus. It is one of the most important elements in personal defense; for its services, there is no anatomical substitute. The stapes grows with dramatic speed, attaining its zenith twenty years before the humerus, or any other long bone, has achieved maturation. The stapes does not resort to the use of epiphyseal centers for growth; it need not be a giant to be important. Delicacy and mobility are required for the discharge of its physiological duties; to that end the stapes has thrown away half of its osseous self and its very marrow. It is the anatomical patriot *par excellence*.

303 E. Chicago Ave.

LXXXIII

FUNCTIONAL APHONIA

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Philadelphia

and

Francis J. Braceland, M.D.

Chicago

The advent of the newer concepts of psychosomatic medicine and the scientific study of the various bodily changes that accompany diverse emotional experiences prompt the laryngologist to reexamine some of his former beliefs. The former arbitrary division of illnesses into the categories functional versus organic can no longer be held for we now realize that all disease processes have both psychic and organic components. Clinicians as a group have been reluctant to admit that psychic factors could have an important bearing on a patient's symptoms and they have been content to relegate this part of the problem to the domain of the philosopher. Heretofore unless we have been able to inspect, palpate, percuss and auscultate the problem presented, we have preferred to believe that it did not exist and yet all of us can recall our singular lack of success in dealing with such problems as functional aphonia. Somewhat reluctantly, therefore, we are forced to conclude that in dealing with our patients we are continually practicing somato- and psychotherapy. Naturally a paper which discusses a subject such as functional aphonia cannot be highly scientific for the subject of which it treats is neither scientific or intellectual, nevertheless, there is no denying that the condition exists.

The problem of functional aphonia with an emotional background is probably as old as man. The scriptures and ancient literature abound with examples of individuals who were "stricken dumb". The subject is of particular interest at this time, not only in civilian practice where its examples have been plentiful due to unrest, insecurity and the "war of nerves", but also on numerous occasions in the ranks of our armed forces. The records of the last war are replete with cases of hysterical amnesias, aphonias, dysphonias, etc., and there is no reason to believe that the same thing will

not happen in this present conflagration. This in itself would seem to be reason enough for an examination of the principles which underlie functional laryngological disorders many of which are in reality the conversion symptoms of an ordinary garden variety of hysteria.

The literature on the subject of functional aphonia is noteworthy for its sparsity and for the divergent opinions expressed. Barlow[1] called attention to the fact that hysterical aphonia was an escape mechanism the result of subconscious conflict which temporarily solves the patient's problems. He states that two other possible solutions would be mental disease or suicide. He insists that hysterical aphonia is not associated with any organic changes and stresses the importance of taking a good history. Stern[2] differentiated the various forms of functional disturbances but in his efforts to simplify the problem, his explanations become confusing. Also he fails to define clearly the terms organic and functional. MacLeod[3] attempted to classify the dysphonias in order to determine which group should be treated by means of faradic current and which group treated by exercise designed to relax tension.

Dundas-Grant[4] attempted to utilize the observations of Negus who stated that in certain animals the efforts of the forelimbs are accompanied by an approximation of the vocal cords. Grant has his patients resist the pressure of a strong spiral spring and restores the patient's voice by involuntary adduction of the cords. His explanations are not particularly convincing. Marsh[5] discussed 16 cases and quoted four other authors warning us that functional aphonia may be associated with organic disease which in itself seems to be a contradiction of terms. While we realize that functional symptoms can be superimposed upon organic disease, it is obvious that an illness cannot, at the same time, be both functional and organic. Oaks, Merril and Oaks[6] reviewed the literature and discussed two cases of recurrent hoarseness and aphonia. They concluded that certain of these cases are due to ethmoid disease.

MacMahon[7] discussed manipulation of the tongue and thyroid cartilage in an effort to overcome muscular resistance. Weiss[8] found fault with the term functional aphonia. He believed that it is a negative term and that it is inadequate. He states: "In functional disturbance, the pathology lies in the function of the organ which if used properly, would perform physiological actions." Zerffi[11] believes that functional vocal disturbances are directly caused by the failure to inhibit the action of the extrinsic or swallowing muscles during the production of the voice. The failure is due to faulty

voice habits carried over since childhood. He suggested that the treatment should consist of a therapy developed by a close cooperation between a laryngologist and a teacher of voice culture. Levbarg[12] recognized the psychic origin of the functional speech disorders and called attention to the fact that hypnosis is a potent weapon in their treatment. Wyatt[13] discussed the relationship between voice disorders and personality conflict. She stated that the treatment of functional voice disorders should be the same as that of any organ neurosis and called attention to the fact that the voice, like any other organ, can be used to express conflict. Her ideas of treatment are along the lines of the Freudian psychoanalysts.

Greene[11] described a voice condition which strikingly illustrates the influence of psyche over soma and in that way it is related to our presentation. In the condition described by him, the patient complains of "a sensitiveness of the throat"; his throat feels tight and constricted and he has uncontrollable choking sensations when attempting to speak. He also has symptoms of paresthesia, radiated pains in the head and neck, excessive dryness of the throat or mouth or in some instances, just the opposite, excessive secretions. Because of its localized manifestations and since it seems to affect people who do a great deal of talking, it was erroneously thought to be due to fatigue and was called phonasthenia. Greene, recognizing the need for differentiating true phonasthenia from the above pseudophonasthenia and recognizing the psychic factors influencing the latter, aptly named the condition psychophonasthenia. He considers the psychophonasthenic as a variant of the hysterical personality. In this we concur and it is our belief that psychophonasthenia is a partial defect, a way station as it were, on the road to hysterical aphonia which might be considered the end of the line.

The mechanism underlying the functional aphonias will necessarily differ according to the problem involved and the individual concerned but for the most part the pattern is not hard to discover. The trouble usually originates in an incompletely recognized conflict between the sense of duty, the desire for human respect and the overwhelming desire to escape from difficult situations.

In the hysterical personality, the reaction is a complete or "all out" one, the patient suddenly loses his voice completely and his problem is temporarily but pathologically solved. The neurasthenic individual manifests a partial reaction; he complains of pain, manifests hoarseness or a plethora of other symptoms. His complaints are more indefinite and he is more prone to talk about them. The hysteric merely transfers his emotional difficulties grossly into the

physical or somatic plane, the symptoms are clear cut and definite and correspond to the individual's idea of how disease processes operate. The hysteric always appears to be less conscious of his problem than is the neurasthenic, he simply appears with aphonia, a paralysis, etc., treats the disability as a detached phenomenon and does not seem to be concerned about it. As long as he retains his symptoms his emotional problem is effectually repressed.

A large proportion of the functional aphonias encountered in soldiers in the first World War followed upon states of anxiety based on long-standing feelings of insecurity. These anxiety states were frequently initiated by physiologic disturbances, the bodily accompaniments of fear or panic reactions. Later they became the expression of true mental anxiety, a sort of state of suspension between the instinctive urge to escape from danger on the one hand and the demands of duty on the other. The transition to an hysterical reaction is made by mental operations unconscious in character and a pathologic solution to the problem is reached. In this manner the patient has accomplished his desire to avoid danger and by the same token he cannot be accused of cowardice. Obviously a soldier who is aphonic must be sent to a hospital for examination and care and thus by this simple means a man who has been exposed to extreme danger escapes from it through no apparent fault of his own and temporarily he is safe.

Whenever we encounter hysterical aphonia we should ask ourselves what difficulty is being solved in this temporary and unsatisfactory manner. Our task is to determine how the aphonia is getting the patient out of his difficulty. The successful solution of this problem is the first step in the treatment of the patient. A brief resumé of several cases would illustrate the mechanisms involved.

CASE 1.—J. C., aged 18 years, female, telephone operator in a hospital. On two occasions the superintendent upon failing to get an answer on signalling the switchboard found the girl unable to talk. The third time she became aphonic was on the night of an important meeting of the board of hospital managers. Careful laryngological examination revealed no organic disease or inflammatory process. Inquiry into her past personal history disclosed that she had never had any previous difficulty. She was attractive, physically healthy and of average intelligence. She had been a satisfactory worker and was apparently anxious to retain her position. The girl's economic situation was poor and she had to support her father and mother. Her working hours were from 4:00 to 11:00 P. M. Investigation disclosed that some time previously she had met a boy who displayed some interest in her. Recently he had secured a new position and his working hours were 8:00 A. M. until 4:00 P. M., and it seemed that her romance was at an end inasmuch as she was off duty only one Sunday a month. She could not quit her job, work was at a premium, her financial support was needed and

she was expected to carry on with her work. Unconsciously she was solving the problem by means of aphonia; a telephone operator who is unable to talk on important occasions is obviously of little value. In her own eyes and in the eyes of her parents and friends, she was a victim of circumstances, she could not continue with the position she claimed to like because of illness. Thus her ego was preserved yet her desires were accomplished. Transferring her to another position in the hospital on the day shift put an end to her difficulties, the symptoms disappeared but her personality was damaged. Having thus accomplished her purpose in a pathologic manner there was a possibility that she would meet other unpleasant situations in the same manner later on in her life.

CASE 2.—M. R., a 15-year-old school girl, attractive and intelligent, of good family background, awakened one morning and found that she was unable to talk. Careful examination revealed no organic disease. On close questioning it was found that her most pressing problem was an intense dislike of Latin and an inability to master it even though it was a requisite for graduation in her school. She was unable to avoid her difficulty by changing her course because Latin was a required subject in the nursing school which she desired to enter. Illness would have saved her face; she would have had to leave school and thus avoid her difficulty and it would not appear to be her own fault. By means of suggestion and an explanation of the problem her voice returned within twenty-four hours. We are aware of the fact that the problem was much deeper than she realized and than it appeared to be, but the solution of the deeper difficulties is the function of the psychiatrist. Our part of the problem was to remove the symptoms quickly, and to give her some insight into her difficulties.

Usually there is but little difficulty in seeing through the smoke of these simple hysterical reactions. They must not be confused with malingering. These patients were not pretending; the transition of emotional problem to physical symptoms was made unconsciously in a personality which to begin with was loosely integrated. Not all of the cases are so simple as these. The phonatory difficulties in the anxiety states and the neurasthenics are not quite so primitive and their treatment is usually a problem for the psychiatrist. We should also think of the possibilities of early dementia praecox in the more bizarre form of functional aphonia which persists over an unduly long period of time.

Mirror laryngoscopy commonly reveals a normal larynx. The vocal cords may be pale and often appear thin. Deep inspiration will be followed by slight abduction of both cords. On asking the patient to say *ah-ah-ah* or *eh-eh-eh* the cords often meet momentarily in the midline and then separate so that as air is forced between the vocal cords practically no sound is produced. It will be observed that immediately after attempted phonation if the patient is asked to take a deep inspiration the cords again separate indicating that both are mobile.

To ascertain if the cords can meet in the midline the patient is asked to cough while the mirror is held in the pharynx. It will be noted that the vocal cords meet normally in the midline and cough sounds are normal. In patients who fail to cooperate instillation of a few drops of normal saline solution with a laryngeal syringe into the larynx by mirror laryngoscopy will very promptly produce cough or spasm of the larynx and adduction of the cords will be observed.

It has been the experience of every laryngologist who has seen patients with laryngeal stenosis, paralysis or other conditions which interefere with proper approximation of the vocal cords, that a loud sound usually can be produced on phonation. The functional aphonic case cannot be induced to speak above a faint whisper irrespective of volitional effort. Cough is normal and there is no dyspnea.

The diagnosis is based on the sudden onset, whispered voice, often a history of previous voice loss, the laryngoscopic appearances and absence of any demonstrable evidence of local disease.

It is important to secure a careful history and to make a complete physical examination, including neurological studies and necessary laboratory procedures, for loss of voice may be observed in cases of prolonged severe illness such as tuberculosis, and following overexertion and excessive use or abuse of the larynx. Examination of the larynx in these cases usually reveals approximation of the vocal cords during attempted phonation in spite of the patient's inability to produce a normal or loud sound. Malingering must not be confused with hysterical aphonia. The hysteric is quite indifferent. There is little difficulty in securing a satisfactory view of the larynx as patients with functional aphonia will cooperate very well. This is in contrast to the malingerer who is not anxious to have his larynx examined, especially during attempts at phonation as he is afraid that something will be discovered which will uncover his fraud.

We have been unable to secure any outstanding diagnostic assistance from roentgen studies of the larynx in these cases.

The methods of treatment about to be discussed are clinical applications and, therefore, are not necessarily to be regarded as scientifically controlled procedures. They are not entitled to scientific credit and by the same token are not to be subject to scientific criticism. As with all empiric measures their value should be judged by their therapeutic efficacy or by their failure. The treatment

actually starts at the moment when the patient walks into the laryngologist's office and each subsequent step has an effect on the patient's illness whether for better or worse. At the risk of appearing to be dogmatic we now offer what can be termed Axiom 1 in the treatment of functional illness; i. e., we should never make a diagnosis of functional disease or neurosis by exclusion. In addition to absence of organic or inflammatory disease, we must have some positive signs of neurosis. This latter positive evidence of neurosis can only be determined by means of the expenditure of some time and a sympathetic understanding and questioning of the complainant and his or her relative.

It is of course necessary to treat and correct any pathologic change which has been found in adjoining structures but at the same time one should realize that the correction of a moderate deviation of the nasal septum or local treatment of a normal larynx will not cure a neurosis. Any unnecessary manipulation or any form of meddlesome surgery simply fixes the neurosis and causes the patient to concentrate his attention upon the part being treated and wrongfully to regard it as the cause of all of his difficulty. Perhaps it would be wise to mention several other things which should not be done.

1. Do not become angry with these patients. Even if they have no demonstrable organic lesion they are ill and often are suffering more than patients who have actual structural lesions.

2. Do not dismiss these patients with some trite remark like "it is all in your head." Do not tell them "to snap out of it."

3. Do not delve too deeply into their psychologic difficulties unless you are prepared to handle any eventuality which might arise. Superficial inquiries are desirable and are likewise perfectly safe.

Irrespective of the underlying psychopathology the relief of symptoms is always important. One should remember, however, that the relief of symptoms in many of the cases is not tantamount to cure. It is only the more superficial types of psychotherapy which are available to the laryngologist. He has neither the time nor the training to indulge in the various forms of deep analysis. While we are cognizant of the fact that the superficial forms of therapy are hardly ever productive of a deep and lasting cure, nevertheless, they do enable the patient to gain some insight into his condition and usually help him to continue his daily life under his own momentum.

Perhaps the most readily available type of psychotherapy open to us is that of suggestion. We all unwittingly use suggestion in our practices and all of us are influenced by it in our normal lives. Suggestion means the giving of an idea or proposition to a patient and having him accept it when there is no logical reason for his doing so. In this way suggestion is said to differ from persuasion, another form of therapy in which the patient is given an idea and persuaded to accept it because logical reasons or instructions are advanced at the same time. Some of the factors which influence suggestion are: (1) the younger the patient the more suggestible he is found to be; (2) the less a person knows about the subject under discussion the more suggestible he is found to be; (3) the third factor concerns the authority with which the patient invests the physician in his particular field of medicine. In this regard it is apparent that the work of a well-known laryngologist concerning dysphonias and aphonias is much more readily accepted than would be the word of the psychiatrist who is unable even to examine the larynx skillfully; (4) the fourth factor concerns the manner in which the suggestion is given. Usually tact, interest, understanding and kindness are necessary. Nothing is ever accomplished by brow-beating the patient.

Therefore, the procedure thus far encompasses:

1. Making a definite diagnosis.

2. Finding the cause of the emotional difficulty whenever possible.

3. Drawing the analogy so that it is evident to the patient without too much detailed explanation and without forcing the interpretation upon him.

4. Using the application of suggestion, keeping in mind the factors mentioned above.

5. The return of function is then attributed to the patient's efforts and not to those of the physician.

The above procedure will be found efficacious in most of the cases but there are others which will not respond. Again we should call to mind the fact that the aphonia like all neuroses is the patient's compromise with his existing circumstances. It is his manner of saying no to life. If he were to give up his symptoms so easily he would be once again faced with the original problem which must have seemed to be more disastrous than the aphonia. If superficial

suggestion will not help, we may then try the next step, namely, deep suggestion, i. e., hypnosis.

To mention the use of hypnosis before a scientific body is to risk possible ridicule but nevertheless hypnosis has been found to be an effectual adjuvant in the treatment of functional diseases. It is highly probable that this war will see its spread as a therapeutic weapon in the treatment of men at the front who, upon displaying signs of functional disease, require some quick and readily available type of efficacious treatment. It came into prominence in the last war and assumed respectability in the treatment of hysterical paralysis, blindness, tics, aphonia, etc. In the last analysis, hypnosis is nothing but deep suggestion administered to a person known by virtue of the nature of his illness to be highly suggestible. It would probably be better if the term "complete relaxation" were used in place of hypnosis because of the latter's unfortunate connotations.

Most of the sufferers from functional aphonia are susceptible to hypnosis. It is useful in the following way:

1. Hypnotically induced sleep is in itself useful as a healing factor.

2. The suggestion given in hypnosis is directed outright against the psychic or physical symptoms which are to be eliminated.

3. Forgotten experiences which may be causative agents in the neurosis are brought back to consciousness.

The procedure is simple; the technique is as follows: After the decision to use this form of therapy has been made the physician explains to the patient what he is attempting to do and why; the word hypnosis need not be used. The patient is usually made to recline in a room removed from noise and distractions. Because of the possibility of complications it is best to have a third party present. The patient's gaze is fixed upon an object held in such position as to tire the patient's extrinsic ocular muscles as he fixes upon it. After the patient's eyes have closed or have been closed, somnolence and relaxation are suggested to him in a monotone. This is continued until the patient is put into a light sleep. The depth of the sleep can be regulated by the physician; in most cases a superficial form of hypnosis is all that is required. After the desired level of somnolence has been reached questions can be asked, suggestion implanted or posthypnotic suggestion can be given. The patient is awakened by means of a prearranged signal.

Failure the first time should not discourage the physician; it is necessary for him to have confidence in himself, however, for any timidity is unconsciously communicated to the patient. The procedure can be repeated several times weekly if necessary. Used with necessary caution there is no danger attendant upon this form of therapy; on the contrary much good can be accomplished. If the patient fails to respond to either suggestion or hypnosis it is probably best to secure psychiatric assistance. Some of these refractory patients are potentially schizophrenic and their failure to respond to superficial therapy is in itself a danger signal. The most easily treated of our patients is the group of conversion hysterics. Those with the mild anxiety states are also readily helped. The psychasthenics are more difficult to treat and will probably require a deeper form of therapy than we are able to manage.

The use of the various forms of physical and electrotherapy in the treatment of functional dysphonias should only be that of accessories. While using them the patient should be made to understand that they are only adjuncts to the main therapy; namely, that of clearing up the emotional background of the illness. If the patient is led to believe that the high frequency current and other similar agents are in themselves curative, he is prone to fix upon them, requiring their use more often and finally to become discouraged, because the condition tends to recur.

A word of caution should be inserted here about local treatment of laryngological conditions which are patently of psychiatric origin. There is a tendency on the part of some laryngologists to treat these patients with instillations and instrumentation in the vague hope that they can make the patient "snap out of it." It should be understood that any small amount of benefit which appears to accrue from these manipulations is due to the suggestion which is given at the same time. These proceedings are not entirely without danger for frequent local treatment in the case of neurotic disorder only serves to fix the neurosis at that particular site and to make later treatment more difficult. These symptoms are the patient's compromise with reality; if by means of a semidramatic treatment, we are able apparently to "cure" these symptoms, the patient will soon be down with symptoms involving some other organ system. Eventually most of these patients end up in the hands of the psychiatrists and their work is handicapped when the patients insist there must have been something physically wrong or else the doctor would not have continued to treat him with medicines and electricity.

CONCLUSIONS

1. Functional aphonia is an hysterical conversion symptom and constitutes an escape mechanism in a personality which is loosely integrated to begin with.

2. The importance of ruling out organic disease before considering functional disorders is stressed. Not only should there be negative evidence of organic findings but also positive signs of the hysterical reaction type.

3. The accompanying voice changes are not due to local laryngeal disease. Mirror laryngoscopy reveals a normal larynx. There is failure to maintain proper adduction of the vocal cords during phonation.

4. The laryngologist should recognize the essentially psychiatric foundation of these conditions and either should equip himself to deal with them or see that the patient is placed under expert psychiatric treatment.

5. The dangers of "fixing the neurosis" by means of protracted medication and instrumentation are considered and attention is directed to the fact that mere removal of the superficial symptoms does not mean that the patient is cured. Usually the only result of treatment by electricity and gadgets is changing of the locus of the complaint.

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LXXXIV

CYSTS OF THE FLOOR OF THE MOUTH*

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During the past twenty years no paper on this subject has been presented before this Society and textbooks on otolaryngology devote very little space to its consideration. Laryngologists are frequently consulted regarding the diagnosis and treatment of abnormalities in the floor of the mouth, but judging from the accumulated literature it seems that most cases are being handled by oral and dental surgeons. In more than fifty articles reviewed, the majority were from European sources and contributed chiefly by stomatologists and dentists.

The multiform nature of cysts and their various ways of developing are of interest. Some of them grow on or in the floor of the mouth, while others become, only secondarily, cysts of this region. Some are true cysts, while others should be considered ectasias of obliterated ducts, results of inflammatory processes or parasitic products. Stein[41] enumerates various types of cysts which may all be observed in the floor of the mouth: ranular, dermoid, epidermoid, thyroglossal, branchial, multiocular, mucous, hydatid, and cystic ectasias of the salivary ducts.

There seems to be no satisfactory classification available which includes all possible cysts of this region. It may be said, however, that they are either congenital or acquired. The congenital cysts are developmental errors. The sublingual and the submental or mental regions which form the floor of the mouth are built up from so many embryonal structures that deformity is likely to occur in regions of such intense activity (Roper-Hall[34]). The entrance to the ectodermal primitive mouth is formed by the frontal maxillary and mandibular processes (first branchial arch). Below this entrance is the mesobranchial field between the mesial ends of the second, third and fourth branchial arches. The structures of the neck develop from these arches and from the external depressions between the arches. After the hyoid is formed we find a sinus below it

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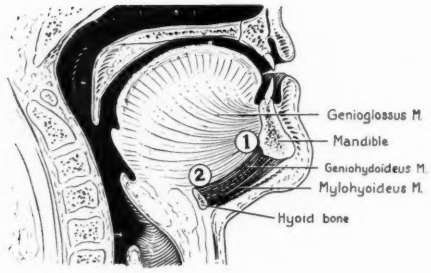

Fig. 1.—Locations of median dermoids in floor of the mouth (after Butlin). From Blair, Vilray, "Surgery and Diseases of the Mouth and Jaws," C. V. Mosby Co., St. Louis, 1912. Used with permission.

which is called the cervical sinus. This later becomes the cervical duct which is connected with a complicated system of epithelial chambers. Normally these chambers will disappear. It may happen, however, that a portion of them is left behind, from which cysts and fistulae may develop. Part of the epithelium may be pushed into the depths, especially in the midline where the mesial ends of the right and left arches grow together. The displaced epithelium retains the vitality of embryonal life and it may at any time develop into cystic formations.

The cephalic intestine also contributes a part in the formation of the floor of the mouth; it is lined with entodermic epithelium which undergoes many changes during embryonal life. The complicated histogenesis of the lining epithelium explains the fact that the congenital aberrations of the epithelium may show all types of cells, including stratified, ciliated, ordinary squamous cells, and cells in mucoid degeneration. Compared with the great frequency of cysts originating from the dental system, cysts of the floor of the mouth are rather infrequent (Willinger[49]). They are all characteristic, being of rather slow growth, and are mostly round or ovoid in shape. Their size depends chiefly on the carelessness of the patient. The subjective symptoms depend on the size. There may be difficulty in speech, mastication, swallowing, and even deformity of the neck or the mandible. The larger cysts may interfere with normal dentition; some of them may cause a marked swelling in the submental region. When infected, painful abscesses may result. Dermoids are especially prone to push the mandible and the dental

alveoli outward or to cause a pressure atrophy of the jaw (Meister[27]). Rarely are they allowed to grow sufficiently large to cause obstruction of the respiratory passages.

The rarity of these cysts accounts for many errors in diagnosis. It is very easy to mistake a sublingual dermoid for a ranula,[49] though their general appearance would make one think that their differentiation is a simple matter. Aspiration for studying the contents is not recommended[27] on account of the danger of infection. Thus it will be possible only after its removal to tell the exact nature by histologic examination. The site of the cyst, e.g., whether it is in the midline of the mouth or lateral, is not sufficient for the recognition of the type being dealt with.

Differential diagnosis from other tumors or pathologic processes of the mouth floor should not be difficult. One should exclude the presence of lipoma, fibroma, hemangioma, carcinoma or sarcoma. Cystic lymphangioma occasionally may be confused with true cysts of the mouth floor,[35] though there are authors who include such tumors of the lymphatics under the general term of ranula. Cellulitis of the mouth floor is easily distinguished from cysts by its rather acute development and its other accompanying symptoms.

Even though the nature of a cyst of the floor of the mouth cannot always be recognized, an incorrect diagnosis is not a particular problem, since all cysts, regardless of their origin, have to be removed surgically.

1. Among the congenital cysts of the floor of the mouth those of the Bochdalek ducts and the thyroglossal duct are mentioned first. These are in fact not cysts of the floor of the mouth; neither are the so-called branchial cysts, but occasionally they may grow so large as to encroach upon this region. Stein[41] reported a case in which a branchial cyst was as large as a hen's egg. Histologically they resembled very much the structure of mucous cysts and dermoids, but their content was different. Neumann[29] referred to them as "ranula."

2. Branchial tumors develop chiefly in salivary glands. They are of epithelial origin and may undergo a cystic degeneration. Such cystic tumors in the sublingual or submandibular glands may appear as cysts of the floor of the mouth.[14]

3. The occurrence of dermoid cysts in this region may be considered relatively rare. Only about 5 per cent of the epithelial cysts of the neck are referred to the first cleft[52] and it is from this cleft

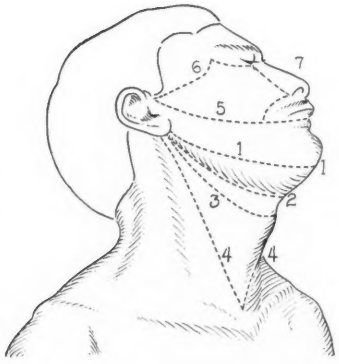

Fig. 2.—Situations of congenital fissures in neck and face. *1*, *2*, *3*, *4*, First, second, third and fourth clefts. *5*, Intermaxillary cleft. *6*, Frontomaxillary cleft. *7*, Nasomaxillary cleft. (From Ewing, "Neoplastic Diseases," Saunders, Phila.)

that cysts of the floor of the mouth proper develop. They are rarely seen on the inferior surface of the tongue. They are then congenital, usually small during childhood, with a more intensive growth at the time of puberty. All cases in my own experience have been seen in patients past 25 years of age. In Kuehns' case[21] the growth of the dermoid became very rapid during a pregnancy of the patient and was so large that she could sleep only in a sitting position. Meister[27] collected 143 cases of dermoid of the floor of the mouth from the literature, and found that in only 3 per cent was the growth so rapid as to necessitate an operation during childhood. In a child the size of the cyst may be that of a cherry, an almond or a walnut, while in adults it may be as large as a hen's egg or an orange. In Putzu's case[31] the patient was unable to close his mouth.

The dermoid cyst is round or ovoid and may be attached to either the mentum or the hyoid bone. The wall is strong, thick, of a yellowish color and the content is that usually found in the dermoid type: hair, other appendages of the skin, lipoid drops, fatty acid crystals, also certain round bodies of a lipoid nature or atheromatous character. The fibrous capsule is lined with stratified epithelium. They are found in both sexes. In the series of Meister[27] 54 per cent were males and 46 per cent females, and the majority required treatment between the ages of 16 and 30 years. A dermoid

cyst may become infected[27] and there is one case on record which resulted in osteomyelitis, sepsis and death. The possibility of malignancy must always be considered. I have one photomicrograph of such a case. An interesting combination of dermoid and ranula has been described by Meister;[27] the dermoid cyst exercised pressure on Wharton's duct resulting in the formation of a ranula-like swelling. According to their site, dermoids of the mouth floor are called lateral and medial. Butlin was of the opinion that they are originally all centrally located and migrate laterally as they increase in size. It is possible then that a dermoid occurring above the epiglottis may have originally had a more forward position. The medial dermoids may be between the geniohyoid or the genioglossus muscles. Putzu[31] classified all inclusion cysts of the floor of the mouth into two groups: (a) those in the median line of ectodermal origin; (b) those derived from the second branchial arch. He also distinguished the mucoid cysts, all of which are of entodermal origin. The medial dermoid cysts may be above the mylohyoid muscle, or below the mylohyoid muscle, or submental. The subjective symptoms produced are different for the two varieties. The sublingual dermoid may resemble a ranula or a retention cyst. If it is large enough it may push the tongue upward and cause difficulty in mastication. The movements of the epiglottis may also be handicapped,[1] and dysphasia and dyspnea may result. Movements of the tongue may be limited and even nasal feeding may be necessary in some cases. The submental dermoid usually causes swelling in the neck[4] and double-chin, which is especially striking in young people.[28] In the case Willinger[49] describes, the dermoid cyst was both sublingual and submental. The mylohyoid muscle divided the two halves of the cyst.

Various hypotheses have been suggested on the congenital origin of dermoid cysts of the mouth floor. Colp,[51] Warren[47] and others assume that the dermoid cysts of this region develop either from the epithelial layer of the tuberculum impar, which is the primordium of the tongue, or from epithelium that became loose at the time of the closure of the first or second branchial arches. Hassel suggested that the dermoids are from the residual epithelium of the vesicula cervicalis, which would explain also the origin of the rare lateral dermoids. Others mentioned a possible trauma of the floor of the mouth in the stage of its embryonal development. Putzu[31] thinks that dermoids originate from mesobranchial tissue. It is probable that they originate from any of the structures mentioned, and Haeffner[12] accordingly distinguishes the possible sites of epithelial maldevelopment: (1) the region immediately behind the median

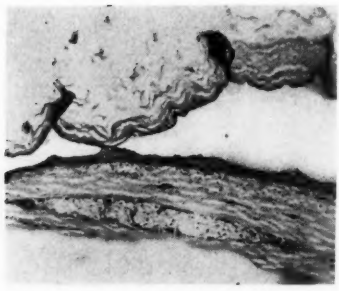

Fig. 3.—Photomicrograph. Dermoid, floor of mouth. The stratified squamous epithelium covering with desquamated cornifying layer is clearly seen with the deeply embedded glands in the relatively thin cyst wall.

position of the mandible; (2) the median position of the hyoid bone; (3) the region between the mandible and the hyoid; (4) the region of the cornu of the hyoid bone.

Dermoid cysts of the mouth floor do not spontaneously disappear, though Behlendorff[2] mentions the possibility of spontaneous regression. There may be, however, an intermission in its growth.[27] The only treatment for this condition is radical removal. Stein[41] operated on three patients through the mouth, yet the majority of authors recommend an external approach. Lee[23] used a modification of the hydrocele operation for a dermoid that was adherent to the mandible. They should be approached according to accessibility, either from the mouth or from the neck.[39] Needless to say, removal can be accomplished more easily when the cyst is small.[38] The use of escharotics to destroy the cyst lining is mentioned only to be condemned.

4. Epidermoid cysts are slightly different from dermoid cysts. Their origin is from the same embryonal structures as the dermoid, but at a later phase of embryonal development when the epithelium of the mouth floor is more differentiated. Karow[19] considers epidermoids as being derivatives of the entoderm. The external appearance of the epidermoids is the same as that of the dermoids, but their contents and the structure of their wall are different. They are lined with an epithelial layer of mucous type[16] and sometimes with ciliated epithelium. The latter type of epithelium is considered a derivative of the thyroglossal duct, and such epidermoids are some-

times called thyrodermoid.[16] Seventy-five per cent of epidermoids are observed in elderly individuals. The content of epidermoids has no hair, no skin appendages or glands. Houpert[16] describes several typical cases: one epidermoid of the inferior surface of the tongue extending to both sides, another multiocular cyst with a central cyst the size of a hen's egg surrounded by several smaller cysts. Recurrence is rare and no report of malignancy has been found.[19] The treatment is the same as that used in dermoids.

5. The region of the floor of the mouth is rich in small and large mucous glands. Occlusion of the orifices of the ducts of these glands may result in cysts, or rather pseudocysts, which have been and are commonly called ranula. Mucous cysts have not been much studied, yet they are rather frequent though of little importance.[42] They may occur anywhere in the mouth where mucous glands are located; only the gingiva and the upper surface of the tongue are free from such glands. These are retention cysts with closed cavities and individual walls which develop from the excretory duct. The glands themselves remain intact, but they may show regressive changes under pressure of the cyst. The wall is very thin and closely adherent to the mucosa and the contents depend on the character of the gland.

According to Take,[42] the first step in the development of a mucous cyst is the closure of the duct of a mucous gland. This closure may be due to injury, inflammation, or inspissation of secretion. This closure is followed by retention of secretion, dilatation of the duct, fusion of neighboring cysts and mucous degeneration of the cells of the dilated duct. Mucous glands are located at various places in the mouth, the most important being the glands of the Blanden-Nuhn on the under surface of the tip of the tongue. Cysts in this region are usually small and resemble the ordinary ranula. Their treatment is total excision of the wall of the cyst. Enucleation is difficult on account of the very thin wall.

6. Polycystoma, or degenerated cystic adenoma, is very infrequent. It is, in fact, a tumor of the mandible but may encroach upon the floor of the mouth.[41] It develops from inflammation of displaced epithelium of the enamel and is mentioned here for the purpose of differential diagnosis.

7. Traumatic epithelial cysts develop from a piece of epithelium displaced and embedded into the depth of the mouth floor after accidental burns, mechanical injuries, or operations on the mouth. The wall consists of connective tissue and the lining is epithelium.

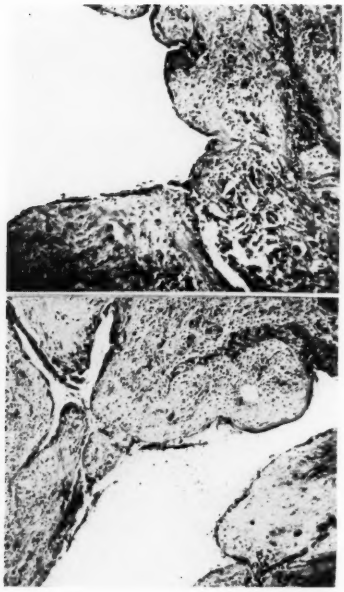

Fig. 4.—Photomicrographs illustrating the loculated character of the bronchogenic cyst showing the compressed, ciliated epithelium and diffuse chronic inflammation with areas of giant cell formation.

They seem to be so infrequent that not a single case has been found described in the literature of the last twenty-five years.

8. Formerly the term ranula meant any cystic swelling of the floor of the mouth. A variety of names has been given to this lesion, all of which indicate a resemblance either to the noise produced by a frog[10] or to its resemblance to the appearance of a frog's abdomen.[41] What cyst of the mouth should be called ranula and what is the exact origin of such ranula are still matters of discussion. Lack of exact knowledge of the ranula explains the rather confused classification of this type of cyst. It is accepted to separate the so-called acute ranula from the ordinarily slowly growing cyst. The former is probably either an inflamed ranula or a cystic dilatation of the duct of the salivary gland caused by a stone.[41] The classic site of a ranula is sublingual, but many submental and submandibular cysts were also described as ranula. It is usually unilateral, but reports of

bilateral occurrences are found in the literature.[20] Prien[30] collected a series of 19 patients of whom 17 were women. It is questionable whether this is of significance. Ranula causes the same symptoms mentioned under dermoid cysts: the tongue may be pushed upward, dentition may be disturbed, the mandible may be deformed, and there may be disorder in swallowing, respiration and speech.[2] The wall of the ranula is composed of quite dense, fibrous tissue. Its content differs from that of a dermoid in that it contains no hair or appendages of the skin.[39, 44] Those who exclude the mucous gland cysts from the definition of ranula say that there is no ptyalin and no salivary element in the content of the ranula.[30, 48] Those, however, who include among the ranulas also the congenital cysts of Wharton's duct assert that there may be saliva in the cyst.[32] By its generally bluish color and clear viscous content, ranulas can be easily differentiated from dermoid cysts.[49] Behlendorff[2] says that malignant degeneration is possible, but is very rare. The theories advanced on the development of ranula are very numerous. Willinger[49] says that it develops from the sublingual gland. Suzanne believed that it was the result of an inflammation of the gland. Others mentioned stomatitis as the cause of obstruction of the sublingual duct and its change into a cyst. Ludwig,[25] however, saw in only one of his 14 patients a preceding stomatitis. Sippel accepts the acquired inflammatory nature and origin, and says that it is mostly seen in people with neglected mouth hygiene. On the other hand, there are many who affirm that ranula is a congenital cyst.[11] Wild[48] says that, in its classic site, it is a cyst of the sublingual gland, while the others cite it as a cyst of thyroglossal origin. Fleischmann described a serous bursa of the floor of the mouth and stated that ranula is a cyst of this bursa. Skillern, in 1919, still believed in this hypothesis.[40] Tsuzuki[45] reported a connection between ranula and syphilis. The writer has encountered one such case which responded to antiluetic treatment. Stein[41] attempted a general definition of ranula and said that it is a degenerative cyst formation of salivary glands in the sublingual region. Ivy[18] was of the same opinion. Camera[7] reported a case of the sublingual type with obliteration of Wharton's duct. Schiemann[35] described a peculiar case of apparent ranula and left the reader in doubt whether the cyst was a primary lymphangioma which had degenerated, or whether it was a detached cystic formation of the duct of the sublingual gland. Thompson[44] is of the opinion that all ranulae are branchiogenetic, developing originally at a lower level and carried closer to the floor of the mouth by muscular action. Hitz[15] suggests the general term epithelial cyst; however, some ranulae are lined with ciliated epithelium, while

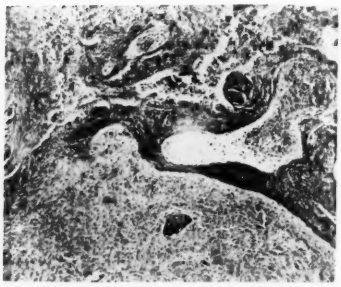

Fig. 5.—Photomicrograph. Malignancy of dermoid. Note in particular the variation in the degree of malignancy found in this one area. The highly undifferentiated squamous epithelial cells with frequent mitoses produce on the inner cyst lining typical acanthomas and epithelial pearl formations.

others may have continuous epithelial lining or all types of epithelial cells may cover its inner surface. This gives rise again to another classification, the ciliated variety and the nonciliated variety. The most important recent study was made by Cottalorda,[8] who, however, stands entirely alone in his views on the pathogenesis of ranula. He concluded that only the ciliated variety of ranula is a true cyst and its develops from the plica frimbriata. The common ranula is not a cyst at all but a pseudocystic tumor without a secreting epithelium and develops from the sublingual tissue of the embryo.

The great differences as to the explanation of the origin of ranula cannot be abolished, however, by a new hypothesis and it remains the task of future histologists and embryologists to solve the mystery. In the treatment it really does not matter whether a cyst of the mouth floor is a true ranula or only a pseudocyst. The best method is to extirpate it in toto.

Many methods of treatment have been suggested, such aspiration of the cystic content, drainage by incision, destruction of the cyst by injecting sclerotic substances into the cyst. Hobert described a special operation consisting of partial excision and eventration of its inner wall and called the method by the fancy name of batrakosioplastics. It is probably the next best method after total extirpation. Goldman[11] recommends a very similar method. Bonnet-Roy[6] combines the above methods with cauterization of the cyst wall.

The total removal may be performed either through the mouth, especially if the cyst is small,[19] or from the neck.[3, 41] The above approaches may be combined if the cyst is both sublingual and submandibular. Blair[3] recommended a permanent fistulization in place of total extirpation if the ranula has large parafaucial extensions. One observer uses a metal fenestrated ring to accomplish this. Camera and Sippel suggested the removal of the sublingual gland together with the cyst, but Laerberg[22] cautions that this may result in dryness of the oral mucosa and predisposition to dental caries. Willinger prefers to open the cyst, evert it and unite the cystic wall to the oral mucosa, creating an accessory cavity in the mouth. Most observers feel that total extirpation when possible is the most satisfactory method of treatment. Ward[46] recommends use of the endotherm knife for opening the cyst and destroying the epithelial lining by the desiccation current. Ferreira[9] successfully used the diathermic fulguration in eight cases of ranula. There may follow various postoperative complications. Local anesthesia, especially if much anesthetic has been injected, may predispose to local edema, which is very painful and may even endanger the patient's life. Willinger[19] suggests the operation should be done under a block anesthesia of the lingual nerve, using as little anesthetic as possible.

SUMMARY

1. The cysts usually found in the floor of the mouth are: dermoid, epidermoid, ranular, thyroglossal, mucous and branchial.

2. The true nature of ranula is still a disputed question and more investigation is necessary to explain its genesis satisfactorily.

3. Laryngologists are often the first to discover or to be consulted regarding the diagnosis and treatment of these lesions.

4. Surgical removal is accepted as the most successful method of treatment.

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LXXXV

IMPAIRED HEARING IN CHILDREN AND SIZE OF ADENOIDS IN RELATION TO SOME ANTHROPOMETRIC DATA AND TO CONDITION OF TEETH*†

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This report is based on observations made in a study of impaired hearing in school children. In the two reports of this study that have been published,[1, 2] the possible relationships of impaired hearing to teeth, stature, nutritional status, etc., have been only briefly mentioned, because these papers considered primarily the major purpose of the investigation; namely, evaluation of irradiation of hyperplastic nasopharyngeal lymphoid tissue with radon as a therapeutic measure to prevent deafness. Relationships of impaired hearing to information, easily obtained in mass examinations of school children, have been sought because such correlations might aid in diagnosis or prognosis and therefore be of practical importance in large scale programs for the prevention of deafness.

The sources of the material and the methods of examination are described fully in the other reports. Briefly summarized, they are: First, in 1939-1940, careful hearing tests and physical examinations of the ears, nose and throat were made of 1365 children. The group is believed to be a truly random sampling of the white children attending regular classes in the third, fourth, fifth and sixth grades of the schools near the Johns Hopkins Hospital. The age range is 8 to 14 years. Second, those children who had impaired hearing and overgrowth of the nasopharyngeal orifice of the eustachian tube by adenoid masses, as determined by examination with a nasopharyngoscope, have been treated with radon by the method described by Burnam,[3] and have been re-examined periodically. Third, children who had tubal overgrowth by large adenoids, but whose hearing was either normal or only slightly impaired for high tones, have been

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re-examined twice yearly. This untreated group is more than double the size of the treated group.

In the present study the hearing tests with which correlations have been sought are those made with a Western Electric Company 1-A audiometer in a sound-proof room. At each examination the thresholds of hearing for 14 frequencies (from 32 to 16384 cycles per second) were determined. To simplify the analysis of the observations, the tones tested are grouped in three ranges; namely, low (32 to 256), middle (512 to 2896) and high (4096 to 16384).

Condition of teeth and acuity of hearing. For this part of the study the children are divided into three groups on the basis of the condition of the teeth at the first examination: good, fair and bad. In the group termed "fair" are included those who had only one or two teeth with caries. To test the relationship of hearing acuity to condition of teeth the first audiograms of the 1365 children are divided into four groups: (1) good hearing bilaterally for all tones, (2) unilateral impairment for some or all of the high tones, (3) bilateral impairment for some or all of the high tones, and (4) one or both ears with impaired hearing for some or all of the other tones, as well as for high tones. For neither boys nor girls is there any significant difference in the distribution of the four types of hearing in the three groupings made as to condition of the teeth: the probability (P), determined by the chi-square test and Fisher's table of significance,[4] is 0.8 to 0.9 for both the boys and the girls. To test the possibility that the condition of the teeth influences the results of treatment, the changes in hearing that occurred while under observation (about two years for each child) are divided into three groups: better, worse and unchanged. For the low and the middle tones (see above) it is definite that the changes in hearing are not significantly related to the condition of the teeth ($P = 0.2$ to 0.3 for low tones, and 0.1 to 0.2 for middle tones). For the high tones a significant deviation from the random distribution is indicated by the chi-square test ($P < 0.01$), but the algebraic signs of the differences in the sub-groups are so irregularly distributed that one must conclude the changes in hearing for high tones also were not influenced by the condition of the teeth. The observations for the untreated children, analyzed by the method used for the treated children, likewise show no significant relationship between the condition of the teeth at the first examination and the changes in hearing acuity during the next two years: for low, middle and high tones, respectively, $P = 0.3$ to 0.5, 0.2 to 0.3, and 0.2 to 0.3.

Condition of teeth and size of adenoids. At the first examination the groups of children with teeth in good, in fair or in bad condition, as defined in the preceding paragraph, had almost the same proportionate distribution of large and of small (or moderate sized) adenoids. There is a tendency for large adenoids to occur more often in children with bad teeth, but the value of P (0.05 to 0.1) does not warrant the conclusion that there is a definite correlation of the two conditions. In the untreated children there has also been a tendency for large adenoids to remain large more often in the children with bad teeth than in those with teeth in good or in fair condition, but statistically the difference is only doubtfully significant. Evaluation of the effect of bad teeth in the treated children on size of adenoids is rendered difficult by the fact that most of the children with bad teeth had dental care as well as irradiation of the nasopharynx. The condition of the teeth, however, does not seem to have influenced the effectiveness of the irradiation therapy either favorably or unfavorably. In this connection attention should be called to the fact that, of the 152 children whose treatment with radon was started during the school year 1939-1940 and who have been re-examined since September, 1941, only 16, or 10.5 per cent, had enough adenoid tissue left this year to require further treatment.[2] Only 3 of the 16 needing additional treatment had bad teeth at the first examination.

Height, weight and height/weight index in relation to acuity of hearing. At the first examinations of the children, standing height was measured to the nearest half-centimeter and weight, with clothing except for shoes and coat, to the nearest tenth of a kilogram. An approximate allowance was made for weight of the clothing worn. The height/weight index used is 100 times the height in centimeters divided by the weight in kilograms. The distribution of the observations for boys and girls separately, at each year of age, shows that these children are fairly representative of white American children, as reported by others.[5, 6, 7]

To determine whether a relationship exists between general nutritional status, so far as it can be measured by height and weight, and the incidence of impaired hearing, the method of comparing distributions in the extreme fourths with each other and with the distribution in the intermediate half has been used. Sex and year of age were considered in determining the sub-groups with respect to physical dimensions. The hearing was divided into four groups, as defined in the first paragraph on condition of teeth. The differences in distribution of hearing types in the tall, the average and the short

children and in the thin, the average and the heavy children are so slight that it is obvious, without further calculations, that no systematic relationships exist. The changes in acuity of hearing that have occurred while under observation likewise show no relationships to height or weight, either in the treated or in the untreated children.

Height, weight and height/weight index in relation to size of adenoids. By the method of analysis described in the preceding paragraph, relationships have been sought between size of adenoids and the anthropometric data. No significant relationship has been demonstrated, either in the entire group of 1365 children at first examination, or in the changes that have occurred in the treated or in the untreated groups.

Shape of face, shape of head, and acuity of hearing. At the first examination of each child an attempt was made, by visual inspection only, to classify the shape of the face into one of Kretschmer's[8] groupings and to classify the shape of the head as brachio-, meso- or dolichocephalic. For neither classification was it practicable, for various reasons, to make the measurements necessary for an exact grouping. Within the crude subdivisions thus made the distributions of the types of hearing acuity (defined in the section on condition of teeth) are so similar that it is obvious neither the shape of the face nor the shape of the head influences the incidence of impaired hearing in children.

Miscellaneous. It was hoped that the observations would make possible an analysis of the relation of allergic conditions to impaired hearing and to the size of adenoids, frequency of "colds", etc. So few of the children, however, gave histories indicative of allergic responses, either to foods or to pollens, dust, etc., that such a study of the data is not warranted. The observations at the first examinations of the children show that only 16 per cent of those with bad teeth are in the "heavy" fourth of the whole material, as determined by subdivision of the height/weight index with respect to age and sex, while 30 per cent are in the "thin" fourth. Sex and age are both important factors in the incidence of impaired hearing, especially for high tones, in children, and are discussed in the other reports.

DISCUSSION AND SUMMARY

It may well be that some easily observed physical or "constitutional" conditions are related to the incidence of impaired hearing in children or to the effectiveness of irradiation therapy of the nasopharynx, and a search for them should be continued. So far as the

present study shows, however, no information of practical importance in a program to prevent deafness in children is afforded by consideration of height or weight for the age, by shape of head or of face, or by the condition of the teeth.

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THE LOCAL USE OF THE SULFONAMIDES, GRAMICIDIN (TYROTHRICIN) AND PENICILLIN IN OTOLARYNGOLOGY*†

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Practically all infections of the ears and sinuses are secondary to an acute coryza or nasopharyngitis. The number of ear and sinus complications seen by the otolaryngologist is growing less each year, due to the widespread use of sulfonamides during the early stages of infection. There are objections to giving large doses of these drugs by mouth and saturating the entire body in order to cure or prevent a local infection. A more satisfactory method of using the sulfonamides to prevent sinus and ear infections is suggested by the publication of Pickrell[1] on the treatment of extensive skin burns. It has long been known that infection is a serious complication of burns, and Pickrell finds it may be prevented by spraying the burned area with a 2.5 per cent solution of sulfadiazine (2 sulfanilamidopyrimidine) in 8 per cent triethanolamine. This mixture has a pH of about 8.7, is not toxic and is readily absorbed by the tissues. To be effective it must be sprayed on the burned area freely and at frequent intervals, at least every hour during the first day, every two hours the second day, etc. Enough of the drug is absorbed under this treatment to maintain a high blood level, but the pre-

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vention of wound infection is probably due to the fact that the concentration of sulfadiazine in the tissues immediately surrounding the sprayed area is six times greater than that in the blood.

This demonstration should be of great interest to otolaryngologists. Much of the discomfort of an acute coryza and the time lost from work is due to secondary infection of the sinuses, ears and lymphoid tissue. Our experience indicates that many of these complications may be prevented by spraying the nasal passages and pharynx with a 2.5 per cent sulfadiazine triethanolamine solution. This mixture is readily absorbed and appears in the blood stream, but since not more than 20 cc. (0.5 gm.) is used in any one day, the concentration in the blood is not high. The concentration of sulfadiazine in the mucous membranes of the upper air passages in these cases has not been determined. After trying this method of treatment on ourselves and numerous patients we became convinced that in order to be effective the nasal passages and pharynx must be sprayed at least eight times a day during the first two days and five or six times a day for the next two or three days. The results were so encouraging that a rigidly controlled experiment was undertaken. The group best fitted for such a study is the training school for nurses. They not only live under the same roof, eat the same food and have the same working hours, but most important of all, they are available for treatment at any hour of the day. All have had a physical examination including the upper air passages, and an accurate record is available of the time lost during the previous year from colds. Partly due to an exceptionally mild winter, only 30 reported for this investigation, some within eight hours and others after three days, but the average was twenty-four hours after onset of acute coryza or nasopharyngitis. No reliable conclusions could be reached unless we knew the exact amount of the sulfadiazine solution used at each treatment and the number and frequency of the treatments. For this reason a special nurse was employed to use the spray on these patients as directed. Nurses with symptoms of an acute cold reported between 8:00 and 9:00 in the morning at a room in the otolaryngological department, where a doctor took the history, made cultures from the nose, nasopharynx and pharynx, a differential count of cells in the nasal secretions and examined the ears and upper air passages. Without their knowledge the patients were alternately placed in "treated" and "control" groups. All were treated exactly alike, except that the controls were sprayed with the

solvent (triethanolamine) and the treated group with the solvent plus 2.5 per cent sulfadiazine.* The routine procedure was:

1. Record the mouth temperature, which varied from 96.8 to 100.2 F.

2. Question the patient specifically about the following symptoms: sore throat, nasal obstruction, malaise, cough and headache.

3. Take cultures: nasopharyngeal secretions were removed with a sterile suction tube and implanted in rabbit blood infusion broth. The broth samples were kept at 37 C. in a water broth until taken to the laboratory. Samples of the broth were streaked on plates of rabbit blood agar and plates were also poured with agar to which human citrated blood was added in order to differentiate the hemolysis of B. hemolytic streptococcus and H. hemolyticus. A swab culture was made from the vestibule of the nose and from the pharynx. Staphylococcus was predominent in the vestibule cultures; pneumococcus, when found at all, was usually in the nasopharyngeal cultures and various types of streptococci were most common in the pharynx. The material for these cultures was obtained with sterile instruments before the nose was sprayed or examined in any way. Cultures from the pharynx were the most reliable and constant. Often a heavy growth was obtained from the pharynx and no growth from the nasopharynx of the same patient. P-aminobenzoic acid (0.5 mg. per cent) was added to all media.

4. Take smears of nasopharyngeal secretions and stain with Wright's stain. A differential count of 200 cells was made. Eosinophiles (20 to 30 per cent) were found in only 3 patients; in all others they were less than 1 per cent. The most striking finding was the relation between a marked increase in the number of monocytes and subjective improvement of the patient's symptoms.

5. Make a routine examination of the upper air passages.

6. Treatment: No patient knew whether she was in the control or treated group. The nose and pharynx were sprayed an equal

*The formula we have used is as follows:

Sulfadiazine powder	2.5 gm.
Triethanolamine	7.5 c.c.
Butyl parahydroxy benzoate "Butoben" (Merck)	0.05 gm.
Boiled distilled H_2O ad	100 c.c.

Boil the distilled H_2O and cool to 90 C. To a portion add the triethanolamine and benzoate. Then dissolve sulfadiazine in this by agitation. Allow to cool, filter and add sufficient boiled H_2O to make up the desired amount.

number of times in both groups. Treatments were at regular appointed hours from 9:00 A. M. to 5:00 P. M. and were not entrusted to the patient. From 1.5 to 2 cc. of the sulfadiazine solution was used at each treatment. Usually eight such treatments were given daily for the first two days and thereafter five or six treatments a day for three days. Subjective improvement usually came suddenly on the second day. Detailed bacteriologic studies showed a decrease in the number of colonies of beta hemolytic streptococcus in the pharynx in the treated group as compared with the controls, but the alpha and gamma strains were unaffected. In addition to the spray a 5 per cent sulfadiazine ointment was applied in the vestibule of the nose twice daily; in the controls, the base was used with no sulfadiazine added. This ointment was used to discourage the growth of bacteria in this part of the nose and to protect the skin from the irritation of secretions. The patients found it very soothing. They were all given a ¼ per cent aqueous solution of neo-synephrin to use when necessary for nasal obstruction. It was rarely used, however, because the sulfadiazine spray is not only soothing, but relieves nasal congestion.

Patients in both the control and treated groups were given aspirin and codeine when necessary for pain in the joints or head, and elixir of terpine hydrate for cough. The most constant and immediate benefit of the sulfadiazine spray was the prevention and control of the irritating cough so often associated with a nasopharyngitis. Not one of the treated cases had a cough severe enough to require terpine hydrate, while spasmodic attacks of coughing from five to fifteen days were a prominent symptom in 9 of the 14 controls. One patient in the control group with a red, edematous pharynx, postnasal discharge and a persistent cough for eight days, was transferred to the treated group, and all symptoms disappeared within forty-eight hours.

Of the treated group 2 of the 16 patients had slight clouding of one antrum on transillumination, which cleared without local treatment, while in the control group 7 of 14 patients had complicating infection of the accessory nasal sinuses. One patient had had acute sinusitis with each cold during the past four years. Six such attacks required irrigation of an antrum during the previous winter. During the past winter the absence of sinus infection is attributed to the use of the sulfadiazine spray.

DISCUSSION

We have made no studies to determine whether or not the use of sulfadiazine as a spray influences in any way the symptoms of

infection with the "cold" virus. It has been shown that a 2.5 per cent sulfadiazine spray, if used frequently enough, will prevent pyogenic infection of a large granulating area following a skin burn. It is not reasonable to suppose that any spray can sterilize the mucous membrane and lymphoid tissue of the upper air passages, but our experience indicates that 2.5 per cent sulfadiazine in 8 per cent triethanolamine is harmless, is soothing to acutely infected mucous membranes, controls the cough and lessens the incidence of complicating sinusitis.

We are not suggesting a cure for the common cold, but a simple method of using one of the sulfonamides to control the growth of pyogenic organisms in the upper air passages, and so shorten the period of disability and lessen the incidence of complicating sinus, ear, pharynx and bronchial infections. The only contra-indication we know of to the use of sulfadiazine as a spray is that some individuals (about 3 per cent) are sensitive to the drug or the solvent and its use causes a vasomotor rhinitis. In contrast 30 per cent or more of the patients taking adequate amounts of the sulfonamides by mouth have nausea, dizziness, fever, skin irritation or some more serious evidence of sensitivity.

Gramicidin, one of the by-products of tyrothricin, was obtained from soil bacteria in 1939 by Dr. Rene Dubos of the Rockefeller Institute for Medical Research. Its value as a therapeutic agent is limited for the following reasons:

1. It is extremely toxic when injected intravenously and there is no evidence that it can be of any use against systemic infection.

2. It is insoluble in water and is not absorbed by tissues. It must be used therefore as a surface application, either in powdered form or in suspension in distilled water or oil. Gramicidin is bactericidal only when in direct contact with the bacterium. It may effectively destroy all gram-positive bacteria in test tube and in animals acutely infected, but gives disappointing results in the treatment of areas chronically infected with these organisms. For example, a small amount of gramicidin suspension injected into the peritoneal cavity of mice will protect against an intraperitoneal injection of 10,000 fatal doses of pneumococci, provided the two injections are made simultaneously. If the gramicidin injection is delayed for a few hours, until the bacteria have penetrated the tissues and become less accessible, the protection is greatly decreased.

3. As the name implies gramicidin is bactericidal for gram-positive organisms. Some types of pneumococcus and streptococcus

are very sensitive to gramicidin, others are resistant. Some stains of hemolytic Staphylococcus areus are the most resistant of all the gram-positive organisms. Gramicidin has little or no effect on the growth of such gram negative organisms as H. influenza, B. coli and proteus.

We have used gramicidin (tyrothricin) in the treatment of more than 100 patients with chronic infection of the accessory nasal sinuses or ears, but it is useless to try to express the results in percentage of cured, improved, or not improved. Results depend on so many factors, such as accurate bacteriologic identification of the infecting organisms, the sensitivity of different strains of the same organisms to gramicidin, the presence of granulations, thick exudate and other conditions which prevent direct contact of the gramicidin with the bacteria, etc. Infected nasal sinuses may contain a variety of bacteria, some sensitive to gramicidin, others resistant. A favorable result must not be expected unless we know that the organisms we have to combat are sensitive to the various agents available for their destruction. In some cases other substances must be applied locally in combination with gramicidin, such as penicillin, which is very effective for staphylococcus, a sulfonamide drug such as succinyl sulfathiazole for the colon bacillus, or active cultures of lactic acid bacilli, which usually cure a proteus infection. Bacteriology is becoming very important and very complicated. If we are to explain our failures to cure an infection, we must not only isolate and identify the various aerobic and anaerobic bacteria, but the streptococcus and staphylococcus as well as the pneumococcus must be typed and the sensitivity of each type to gramicidin, penicillin and the sulfonamides determined by biologic methods.

Our experience indicates that gramicidin is most valuable to the otolaryngologist when used to supplement surgical operation. After the infected area has been cleaned out as thoroughly as possible, postoperative fever, swelling, pain and the necessity for irrigation or frequent dressings may all be prevented by painting the entire operative area (bone and soft parts) with gramicidin and packing the wound with gauze saturated with gramicidin. This result is obtained only when the infecting organisms are sensitive; if they are resistant, the gramicidin treatment has no effect on the postoperative course.

The value of gramicidin has been demonstrated conclusively in patients operated upon for chronic infection of the accessory nasal sinuses or mastoid. At the time of operation the predominating organism in all of these cases was either an aerobic or anaerobic streptococcus, or some type of pneumococcus. The conclusion that

gramicidin was effective is based on the remarkable infrequency of systemic and local evidences of wound infection and the absence of streptococcus and pneumococcus in postoperative cultures. These postoperative cultures are rarely sterile, but now the predominating organism is usually staphylococcus, which as we know is very resistant to gramicidin, and the use of penicillin or some other agent is indicated. Gramicidin is no wonder drug like the sulfonamides, administered by mouth or intravenously in acute infections. Favorable results in chronic sinus and mastoid infection depend on the thoroughness of the operation. The use of gramicidin greatly lessens the postoperative discomfort and shortens the period of hospitalization. It is for these reasons that we think gramicidin is a valuable addition to the armamentarium of the otolaryngologist. It is better than the sulfonamide powders, which in the nose, sinuses and ears form crusts that interfere with drainage.

In the treatment of acute mastoiditis and sinusitis sulfonamides should be given by mouth, but if paracentesis, irrigation of the maxillary sinus or operation is indicated (and it should be remembered that the sulfonamides are of little or no value if an abscess has formed) gramicidin locally is of great value.

The strength of the gramicidin suspension must not exceed 1 mg. per cubic centimeter. Gramicidin comes in powdered form. It is soluble and stable in alcohol. A stock solution of 50 mg. per cubic centimeter in 70 per cent alcohol should be made. If 20 cc. of a suspension is needed for a treatment, 0.4 cc. of the alcoholic solution is withdrawn with a tuberculin syringe and added to poppyseed oil or distilled water. This makes an opalescent mixture and should be used immediately. The oil suspension is preferable when used to saturate the packs; the distilled water suspension for irrigation of the ear or the accessory nasal sinuses. For postoperative irrigation treatments it is better to reduce the concentration to 0.5 mg. per cubic centimeter.

Penicillin cannot be purchased at the present time in the refined form suitable for intravenous administration, but it may be made in any bacteriologic laboratory by growing the mold penicillium on the surface of suitable fluid media.* When the end point is reached,

*Dr. A. M. Fisher of the Medical Staff of the Johns Hopkins Hospital, who has made all of the penicillin we have used, tells us that the media which has been most successful in his hands is the following: tryptone (Difco) 2 per cent; glucose ½ per cent and sodium chloride ½ per cent made up in distilled water. The pH is adjusted to 6.5. This mixture is sterilized by autoclaving and then put into sterile flasks, which are about 14 inches in height and have two sides flattened. The amount of

as determined by biologic tests, the pH and the color, the fluid on which the fungus was grown is passed through a porcelain filter to remove all mold and bacteria. The filtrate is suitable for local application in infected wounds and for irrigation of the middle ear or accessory nasal sinuses. It should be kept sterile and in a refrigerator, preferably frozen. Proteus and some other gram negative bacteria grow freely in a penicillin solution.

Penicillin is superior to gramicidin in that it is soluble in water, does penetrate tissues and is neither toxic nor irritating to mucous membranes. It is especially effective for staphylococcus, but is also bactericidal for aerobic and anaerobic streptococcus and pneumococcus. Like gramicidin, however, certain strains of all these organisms are so resistant that they will grow freely in broth to which penicillin has been added. Here again the intelligent use of this bactericidal agent necessitates careful bacteriologic study of each individual patient.

SUMMARY

There are several nontoxic solvents that will hold as much as 2.5 per cent sulfadiazine in solution and so permit this drug to be applied locally as a spray. For the prevention of infection with pyogenic organisms this method of saturating the tissues in a localized area has distinct advantages over giving the drug by mouth. Administered as a spray it prevents infection of skin burns, and we believe it is the simplest and most logical way to reduce the incidence of infection of the ears and sinuses in colds.

Gramicidin and penicillin are powerful bactericidal agents for most but not all organisms. They usually destroy anaerobic and the Lancefield B. streptococci, a group of organisms resistant to the sulfonamides. To be effective gramicidin must come in contact with the infecting bacteria; it cannot be used intravenously or subcutaneously. Its chief value in otolaryngology is to prevent postoperative

culture media in each flask should be just enough to form a layer 1.5 to 2 cm. in depth when the flask is in the horizontal position. With the flask in this position some of the stock fungus culture is added. The inoculated flasks are kept at room temperature, which should always be between 20 and 25° C.

Usually after seven to twelve days the surface of the media is covered with a thick greenish growth of the fungus (penicillium). Characteristic yellow drops appear on the surface of the fungus growth. The fluid media is quite yellow at the time it is ready for harvesting. It is passed through a Seitz filter to remove all fungi and bacteria. This filtrate contains in solution the substance we call penicillin and is suitable for local application. Its effect on the growth of pyogenic organisms is determined by inoculating a series of 5 cc. broth tubes, using one as a control and adding different dilutions of penicillin to the other tubes. By observing the bacterial growth, a fairly accurate estimate of the potency of penicillin may be obtained.

wound infection. It is almost totally insoluble in water and must be used as a suspension. Applying it freely to exposed bone and surrounding soft parts at the conclusion of an operation for infected nasal sinuses or mastoid, and saturating the packs with a 1 per cent suspension of gramicidin in oil, adds greatly to the postoperative comfort of the patient and shortens the period of hospitalization.

Penicillin is even more valuable than gramicidin. It is soluble in water, is not toxic, and is absorbed by the tissues. It is often more effective than the sulfonamides or gramicidin in combatting staphylococcus infection and is equally effective for aerobic and anaerobic streptococcus and for the pneumococcus. Since most chronic infections in the upper air passages are due to a variety of organisms, the intelligent use of these various bactericidal agents demands accurate identification of the infecting bacteria and biologic tests to determine their sensitivity. Certain strains of streptococcus, staphylococcus and pneumococcus are so resistant to the sulfonamides, gramicidin or penicillin that statistics of their therapeutic value are useless, unless we know with what combination and strain of bacteria we are dealing. We do not wish to discourage the use of penicillin and gramicidin by insisting on these elaborate bacteriologic and sensitivity studies. If used properly both penicillin and gramicidin are harmless, and they should be used freely to help control infection, but if we are to explain the failures to cure we must employ bacteriologic and sensitivity studies.

We otolaryngologists should squarely face the fact that a new era has dawned for our specialty. The sulfonamides, gramicidin and penicillin are only the beginning, but they have worked marvelous cures in many cases which in the past could not have been achieved with surgery alone. Other and even more efficient remedies are sure to follow. The use of roentgen rays and radium in the treatment of certain nose and throat diseases is on the increase. We must familiarize ourselves with all aspects of modern medicine including bacteriology, nutrition in its broadest sense, chemotherapy and irradiation therapy. The otolaryngologist must be prepared to play his part in this new era.

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LXXXVII

THE SULFONAMIDE DRUGS IN THE TREATMENT OF ACUTE OTITIS MEDIA*

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The treatment of acute suppurative otitis media and mastoiditis with the sulfonamide drugs has been of great importance to otologists for the last five years. The development of new preparations, with their advantages and disadvantages, has been so rapid that it has been necessary to keep in constant touch with the research hospitals and laboratories as well as with the current literature. Constant research has been directed toward the development of new formulae which retain or increase the bacteriostatic properties so necessary to successful treatment of the coccal infections and which at the same time prove to be less toxic to the individual.

In the order of their development these drugs are sulfanilamide, sulfapyridine, sulfathiazole, and sulfadiazine. Other preparations are undergoing experimental studies and may prove to be the drugs of choice in the near future.

It is interesting to note the sequence in their development. Sulfanilamide, the active principle of the original preparation, known as prontosil, was the drug of choice in the treatment of streptococcal infections. The toxicity was rated as high. Sulfapyridine proved to be much more effective against the pneumococcus and with a toxicity lower than that of sulfanilamide. Neither of these drugs proved to have much influence over the staphylococcal infections but with the synthesis of sulfathiazole, which is still less toxic than the former two drugs, it proved to be effective against not only the staphylococcus but the streptococcus and pneumococcus as well. The last of the series, sulfadiazine, is the least toxic of all and is effective against all of the coccal infections. Lyons,[1] whom we at the Massachusetts Eye and Ear Infirmary follow, advises the use of sulfadiazine for our purposes in all infections. However, sulfanilamide and sulfapyridine are held in reserve for refractory infections

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with the streptococcus and pneumococcus respectively and the sulfathiazole is preferred for the staphylococcal infections. Finland,[2] writing as late as November, 1941, lists sulfadiazine as first in his "tentative order of preferences of sulfonamide drugs for various kinds of acute bacterial meningitis." The introduction of sulfadiazine is still too recent to allow of any extended reports upon its clinical advantages.

Clinical experience and research over the last few years have established certain definite concepts in regard to the use of the sulfonamides.

I. The sulfonamides are free to act upon infection wherever they can be carried by the blood stream, therefore the most striking results occur in what may be called "free infections" such as meningitis, bacteremia and pneumonia. Wherever the blood stream is free to carry the drug, we may expect therapeutic results. The natural corollary is that wherever the blood vessels are blocked by thrombosis, as in proximity to a localized abscess, the drug is not free to act and one of the primary principles of surgery comes into play, namely, that a localized suppurating focus must be evacuated. This principle is peculiarly applicable to the ear spaces where the blood supply is meager, the vessels small and easily thrombosed and the infection enclosed in a bony cavity.

II. The presence or absence of antibacterial antibodies is important. It is possible for an individual to recover from a streptococcus infection without developing an antibacterial immunity but Lyons[1] has shown that "delayed responses and a tendency to recurrences of the infection predominate in those patients who have no antibacterial antibodies at the time of treatment and who fail to develop antibodies subsequently." Kolmer[3] states that there "is an important relationship between chemotherapy and immunity insofar, at least, as the bacterial infections are concerned"; again, "sulfanilamide and its derivatives are not always a short cut or royal road to success in the treatment of severe infections and physicians cannot afford to neglect biotherapy and adjunct measures which have served them so well in the past."

III. The toxicity of the drugs is well known and we need only to emphasize the fact that it manifests itself usually in the second week of its administration, more often between the tenth and fourteenth days. At times a rise in temperature may occur during the first twenty-four hours and only by stopping the drug will a correct evaluation of the progress of the infection be possible.

IV. It must be recognized that a small percentage of patients, 10 to 15 per cent, become sensitized to the drug. If the preliminary medication has failed to stop the spread of the infection, the re-administration of the drug, in order to combat any complication which may have arisen later, can be accompanied by alarming symptoms such as high temperature, skin eruption, and severe toxemia, thereby causing a severe handicap to the correct understanding of the progress of the disease and its successful termination.

V. Perhaps the most just criticism of the use of the drug in otitis media and mastoiditis is the tendency to mask the symptoms of a progressing infection. This has been repeatedly stressed in the literature. This is particularly important because of the wide uses of the drug in upper respiratory infections and otitis media by the general practitioner and the pediatrician. If this masking of symptoms has made it much more difficult for the experienced otologists to follow the course of an infection, how much more difficult it must be for the less experienced. The situation constitutes a real menace.

VI. It is recognized that there is a definite but small percentage of mortality connected with the use of the more toxic compounds of the group; namely, sulfanilamide and sulfapyridine. There is a universal agreement as to the value of the sulfonamides in the treatment of the complications of otitis media and mastoiditis; the advantages to be gained far outweigh the disadvantages. The criteria for the successful treatment of meningitis and bacteremia with the drugs now at our disposal have been established and generally agreed upon. However, there is no such general agreement as to the treatment of the incipient infections of the upper respiratory tract, including the middle ear and mastoid. The first enthusiasm is giving place to a feeling that the disadvantages may outweigh the advantages. The toxicity, the chance of sensitization, the masking of symptoms and the small but definite mortality offer serious objections to the full therapeutic use of the drugs in all cases of acute otitis media, a disease which most otologists agree resolves without complications in about 90 per cent of the cases.

Otitis media is essentially a disease which tends toward resolution without complications in a large majority of cases following early myringotomy. It is distinctly seasonal, Page[4] finding that 36 per cent of his series occurred in winter, 40 per cent in spring, 15 per cent in summer and 10 per cent in the fall. As a rule the severity of the infection depends upon the virulence of the organism which predominates in the prevailing epidemics.

Epidemics of upper respiratory infections vary greatly in their predilection for involvement of the middle ear and as a rule the severity of the otitis media depends upon the virulence of the predominating organism. Not only do epidemics vary in virulence but they may show a distinct cycle beginning with a mild otitis gradually increasing to a peak of virulence and then subsiding. An early fall or late winter epidemic of acute upper respiratory infections and influenza may be accompanied by a mild or virulent type of otitis media and Kopetzky has reported a distinct periodicity in the virulence of infections and has suggested that a major cycle occurs about once in six years.

The infecting organisms are also subject to considerable variations. Page[4] isolated fifteen different types of bacteria, Henry and Kuhn[5] thirteen types, gram-positive cocci accounting for the greatest percentage of the positive cultures (95 per cent). However, both reports show a surprising similarity in the number of cultures returned with no growth, Page reporting 23 per cent and Henry and Kuhn 23.7 per cent, although clinically all the patients had an acute otitis media, a point to be borne in mind in the treatment.

"Resistance of the patient" is a rather vague term still useful to denote the general constitution of the patient, such as age, state of nutrition, the presence of such chronic disease as diabetes, chronic nephritis, etc., but in the light of present knowledge the term refers perhaps more to the presence or absence of antibacterial antibodies. These are some of the many variables which will influence the judgment of the otologist as to the necessity for chemotherapy or even as to the necessity for myringotomy.

The objective in the treatment of acute otitis media is to obtain a reduction in the incidence of surgical mastoiditis. The question then arises as to whether it is good practice to administer a drug of proven toxicity to its full therapeutic limits in a disease which shows a tendency to recovery in 90 per cent of the cases under well-proven procedures. The full therapeutic use of these compounds has certain implications in regard to toxicity, sensitivity, masking of symptoms and even mortality which cannot be disregarded. It is impossible, therefore, to postulate definite indications for the use of these drugs in such a variable disease as acute suppurative otitis media. The otologist from his experience must judge as to the necessity with due regard to the prevailing factors, such as the seasonal variations, the virulence of the prevailing epidemic, and its manifestations in the individual patient.

Maxwell[6] has outlined a few general principles as a guide for the use of the sulfonamides in otitis media:

1. Acute fulminating infections in the middle ear producing signs of mastoid involvement at the end of the second or third day and characterized by sepsis, profuse aural discharge, pain and mastoid tenderness. In this type of disease there is frequently a spread of the infection by septic thrombophlebitis of the small communicating veins of the mastoid.

2. Acute infections in the middle ear, either mild or severe, but secondary to a virulent process in the throat, nasopharynx, nose or paranasal sinuses. Chemotherapy in these cases may prevent a suppurative mastoiditis by controlling the primary infection.

3. Early, acute, rapidly developing middle ear suppurations characterized by a red edematous tympanic membrane and thin seropurulent or serosanguinous exudate under pressure in the tympanum. This is the type of infection which may spread rapidly to the mastoid if not brought under control promptly.

4. All cases of acute suppurative otitis media from which a pneumococcus is recovered on culture.

The literature of the years 1938, 1939 and 1940 deals with the results obtained from the use of the more toxic drugs, sulfanilamide and sulfapyridine. Many authors (Clein,[7] Lindsay,[8] Horan and French,[9] Bowers[10] Stuart[11]) have reported favorably on the treatment of the acute suppurative diseases of the middle ear by the use of these compounds. Others, such as Babcock,[12] doubt that the decrease in the incidence of mastoiditis is necessarily due to the use of the drug. Lindsay[8] says "the treatment for a patient who is desperately ill is not necessarily the criteria of the best policy in the early stages of those infections or in a moderate or mild type of otitic suppuration."

Many feel that sulfanilamide and sulfapyridine and sulfathiazole should not be used empirically in all acute infections of the middle ear but should be reserved for use in the more severe infections which threaten to involve the mastoid cells. The introduction of sulfadiazine, however, forces a modification of this conclusion. This compound has a distinct influence upon all three of the common coccal infections while exhibiting a greatly lessened toxicity and no mortality, as far as a search of the recent literature reveals. It can, therefore, be administered at the very onset of the infection before cultures show definitely what the invading organism is and

may then be replaced by sulfanilamide or sulfathiazole, if the course of the infection should make it desirable. It is not used, however, without an appreciable degree of morbidity and should only be used for definite indications.

It has been suggested by some that the formation of antibacterial antibodies may be delayed by the early administration of these compounds and, therefore, it should be withheld at the beginning of the infection. A majority, however, advise starting the treatment immediately upon the appearance of the infection, while the blood stream is yet free of thrombosis and the drug can be carried to the seat of the infection.

Lyons[1] has tried to use "a chemotherapeutic blitzkrieg in an effort to conclude treatment in from seven to ten days, thereby avoiding the hazards of prolonged administration." He assumes "that the clinical objective in the chemotherapy of hemolytic streptococcus infection should be to secure dramatic improvement within twelve hours and a normal temperature and an apparently arrested infection within forty-eight hours. Failure to secure such a response demands investigation of the following possible causes:

1. Inadequate blood concentrations of the chemotherapeutic agent.

2. Deficiency of antibacterial antibody.

3. Coexistent erythrogenic toxemia of scarlet fever.

4. A focus of inflammatory fixation; i.e., undrained pus or necrotic tissue.

5. Intravascular or perivascular sepsis, either thrombophlebitis or endocarditis.

6. Drug toxemia.

This procedure is fully justified by the results to which the author can testify but is applicable only to certain selected cases of otitis media because it demands hospitalization and the closest cooperation with the laboratory.

Long[13] has suggested that chemotherapy might be used for 48 to 72 hours at the beginning of an earache in an effort to prevent the development of a suppurative otitis media.

There undoubtedly exists a situation fostered by the confidence of the patient, the general practitioner, the pediatrician, and the

otologist that leads to a half-hearted and wholly inadequate treatment with the drug which frequently confuses the entire picture.

Clinical experience with the drugs would lead one to follow the assumption of Lyons that, if the treatment is to be successful, there will be a marked change for the better within forty-eight to seventy-two hours. We can then draw up a tentative program to be followed in those cases in which, in the judgment of the otologist, the administration of the drug is indicated.

1. Hospitalization, if possible.

2. Confinement to bed during the treatment.

3. Adequate dosage of sulfadiazine to obtain a blood level of at least 10 mg. per 100 c.c. of blood. This will depend upon the age of the patient and the individual's capacity to sustain a satisfactory blood level.

4. An arbitrary time limit of seventy-two hours. By this time the report upon the cultures taken at the time of the paracentesis will be available. If the progress is satisfactory, the drug may be omitted or continued at the same or reduced amounts for a period not exceeding ten days. If progress is not satisfactory, either sulfanilamide for the streptococcus, sulfapyridine or sulfathiazole for the pneumococcus, or sulfathiazole for the staphylococcus should be substituted for the sulfadiazine.

5. In the virulent infections, the prompt determination of the presence or absence of antibodies, and the institution of appropriate measures.

In spite of the administration of the drugs in what is considered to be adequate dosage and the institution of other appropriate measures, mastoiditis will develop in a certain percentage of cases. This may be due to the rapid spread of the infection to the mastoid process when a small focus becomes established and is walled off by a nidus of thrombosed vessels. There is some evidence to show that the middle ear and mastoid frequently become infected at the same time and that it is not necessary for an interval of time to elapse for the infection to spread from the tympanum to the mastoid. This seems to occur most frequently in the acute thrombophlebitis type of infection. It has also been observed that some strains of bacteria are more resistant to the action of these compounds than are others.

From the viewpoint of the otologist it would seem wise to adopt rather strict rules of procedure in the use of the drug in

simple acute suppurative otitis media because with involvement of the mastoid the final responsibility is his. The continuance of a purulent discharge from the middle ear following a week of what according to our present knowledge is considered adequate medication with the drug from the very beginning of the infection should be sufficient evidence that the infection is not controlled. In continuing the drug we assume a grave responsibility in regard to toxemia with its attendant symptoms, sensitization, and a masking of the symptoms of mastoid involvement, while from past experience we know that the ear may continue to drain for from one to three weeks with final resolution and without surgical intervention in the mastoid. Again, the resolution of the middle ear with normal hearing following a week or two of chemotherapy does not relieve us of our responsibility in regard to a possible mastoid involvement. Only too frequently recourse must be had to repeated x-ray examinations to determine the presence of any late infections.

The introduction of the sulfonamides poses new and difficult problems not only to the otologist but to the roentgenologist. In regard to mastoid x-rays in conjunction with sulfanilamide Law[14] says: "The appearance on the film indicates that the mastoid is not as severe as the clinical evidence suggests. The drug does something to the contents of the cells whereby they are made more translucent to the x-ray, so much so that we have had cases of a severity which indicated operative interference and yet, from a single film alone, you could not tell which was the side that was involved."

We have confined the discussion to otitis media which has been treated from the onset with the sulfonamides. What is to be our judgment when confronted by an infection of seven to ten days' duration with signs of mastoid involvement and which has not been treated by sulfonamides at all or at least by markedly inadequate dosage?

The effect which the sulfonamide group will have upon a given infection is unpredictable. Many instances of definite surgical mastoiditis which have not had the benefit of chemotherapy have been reported as cured following the administration of the drug. Here again the time element is important. The benefit should be very marked in from twelve to forty-eight hours and constant vigilance is necessary for several weeks before the patient may finally be considered as cured.

Finally, it is evident that the year 1941 saw a marked falling off in the number of simple mastoidectomies performed. The general opinion has been that ear infections have been very mild irrespec-

tive of the use of chemotherapy. Hospital statistics of the number of simple mastoidectomies performed at the Massachusetts Eye and Ear Infirmary and the Children's Hospital do not reveal a sharp drop until the year 1941, although the sulfonamides have been in general use since 1938. At the Massachusetts Eye and Ear Infirmary the average number of simple mastoidectomies performed during the eleven years from 1930 to 1941 was 246. During 1941 there was a sharp drop to 150, a decrease of 39 per cent. At the Children's Hospital private ward for the decade 1931 to 1940 the average was 61 and for the year 1941 was 27, a decrease of 56 per cent. In wards of the Children's Hospital the average for the eleven years 1930 to 1940 was 93 and for the year 1941 was 94. This is not significant in itself, but in the light of the fact that there were 105 operations in 1939 and 140 in 1940 it assumes more importance.

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LXXXVIII

SULFATHIAZOLE IN MASTOIDECTOMY WOUNDS REPORT OF 84 CASES*†

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The use of sulfonamides in infected wounds has proved, during the past three years, of great value in decreasing morbidity and lowering mortality. A recent impressive account of the value of these drugs can be found in reports of their use on the wounded at Pearl Harbor.[1, 6] Although local implantation of these agents is now a generally accepted procedure, attention must be paid to careful selection of the most suitable sulfonamide.

In June, 1941, Osgood[7] suggested the use of sulfanilamide as an adjunct to mastoid surgery. When the series of cases which I am reporting was started, in July, 1941, I could find no report in the literature relative to the actual use of any sulfonamide in the treatment of mastoidectomy wounds. Since then, however, two reports have appeared—one by Livingston[4] in September, 1941, the other by Guerry and Putney[3] in March, 1942.

Livingston's report gives his results with sulfanilamide in 13 mastoidectomy wounds closed tightly without drain. Primary healing, he found, had taken place in nearly all of these cases, and the postoperative period had been greatly shortened. The average healing time for all 13 wounds was ten days. Eleven of the 13 mastoidectomies he classified as successful. In these the average healing time was 6.4 days. Prior to this series he had used a rubber wick drain together with the sulfanilamide. The wounds in which a drain had been used showed no improvement over those in which no drug had been used locally.

Guerry and Putney reported the use of sulfathiazole in 20 simple mastoidectomy wounds and compared the results obtained in these cases with those in 20 control mastoidectomy wounds in which

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no sulfathiazole had been used. In addition to the insertion of sulfathiazole powder they had packed the wound cavities with one-half inch plain or iodoform gauze which was not disturbed for five or six days. They found that the "average time required for healing was shortened by 16.7 days, or reduced by slightly more than one-half" by the use of sulfathiazole. Furthermore, in the cases in which this drug had been used there was 52.8 per cent less drainage, and the postoperative hospitalization was 55 per cent less than that in the cases in which sulfathiazole had not been used. They concluded therefore that healing was promoted by the use of sulfathiazole.

Histologic studies conducted on laboratory animals by Taffel and Harvey[9] showed no evidence of poor or retarded wound healing attending the use of sulfonamides. On the contrary, they believed healing was actually accelerated in the majority of their experimental wounds.

In the ten months from July 1, 1941, to May 1, 1942, 84 mastoid operations were carried out at the University of Oregon Hospitals and Clinics, 70 of which were performed by me. Twenty were radical procedures and 64 simple. In 74 instances the operation was done by the postauricular route and in 10 instances by the endaural route. Complete simple, or radical mastoidectomies were done in the classical manner in all the postauricular operations. Care was taken in each instance to remove all the cells, and an attempt was made to carry out the same technique and to observe the same degree of thoroughness in each operation. The only variation was that in some cases a soft rubber wick drain was placed in the inferior angle of the postauricular incision and in others the postauricular wound was closed tightly without drain.

The technique was, briefly, as follows: Powdered sulfathiazole crystals were sprinkled into the depth of each mastoid wound and gently massaged into the soft tissue. Care was taken to use plenty of the powder, but only enough so that it would become thoroughly moistened with the blood and the tissue fluids in the wound. The amount varied from an estimated 1 to 3 drams, depending upon the size of the operative defect. When the dressings were subsequently changed, additional sulfathiazole powder was blown over the incision.

So far, only two of the patients have been operated upon a second time. In neither instance was there any evidence of retained sulfathiazole following the initial operation. In one instance, however, the wound edges separated after the second operation, disclosing the presence of sulfathiazole within the depths of the wound.

The residual sulfathiazole in this case was partly washed out on two or three occasions, after which the wound healed without further interference. It was concluded that too much sulfathiazole had been used at the time of the second operation.

In addition to the local implantation of sulfathiazole, the drug was given orally before and after operation to all patients with acute mastoiditis and to some with chronic mastoiditis. The usual oral dosage was 1.5 grains per pound of body weight. Routine oral administration of a sulfonamide, in usually accepted dosage, before and after operation will, I believe, improve operative results.

In order that the results of the present study may be accurately compared with those of Livingston and of Guerry and Putney, who reported on the use of sulfonamides in simple, postauricular mastoidectomies, only the unselected, postauricular, simple mastoid operations were chosen for comparison. Of the 52 operative wounds in the present series selected for comparison, 28 had been closed with a rubber drain. In this group of 28 wounds closed with a drain, the average hospitalization was 17.4 days, the average postoperative hospital stay was 12.4 days, and the average postoperative wound drainage was 20.3 days. The remaining 24 wounds had been closed tightly without drain. Following this treatment of the wound, the average hospitalization was 15.6 days, the average postoperative hospital stay was 8.9 days, and the average postoperative wound drainage was 8.2 days.

Listed in the accompanying table are the results obtained by Livingston, by Guerry and Putney, and by my associates and me. The best results, as is readily seen, were obtained in the wounds which had been closed tightly without drain and abundantly sprinkled with sulfathiazole. These figures, however, tell only part of the story. The most striking result was that almost all of the wounds healed by primary union with practically no drainage and that they had a firm, healthy pink appearance. It was held necessary to record as drainage even the slightest spotting on the dressing. This, therefore, accounts partly for the fact that the postoperative drainage was as high as 8.2 days. Also, the average postoperative hospital stay could have been shortened had it been possible to send every patient home as soon as he had recovered sufficiently to make discharge from the hospital safe. But most of the patients operated upon came from outlying sections of the state. And many of these patients could not be sent home as soon as desired because adequate care would have been impossible if they had returned to their homes.

COMPARATIVE RESULTS OF SULFONAMIDES IN SIMPLE MASTOIDECTOMY WOUNDS

Author and Manner of Treatment	Cases	Average Days Postop. Drainage	Average Postop. Hospital Days
LIVINGSTON			
Sulfanilamide without drain	13	10.0	----
GUERRY AND PUTNEY			
No sulfonamide with drain	20	31.6	8 - 85
Sulfathiazole with drain	20	14.9	6 - 20
CURRIER			
Sulfathiazole with drain	28	20.3	12.4
Sulfathiazole without drain	24	8.2	8.9

It should be noted that the results of the present survey were obtained in entirely unselected cases. All operations for chronic mastoiditis were included. In fact, these comprise by far the majority of cases in the present series. Livingston reported only the results of operations for acute mastoiditis. It is well known that it is much more difficult to obtain a nondischarging wound after operation for chronic mastoiditis than it is after operation for acute mastoiditis. If, therefore, only operations for acute mastoiditis had been selected for the present study, the results would have been even better than those here reported.

Experimental studies have shown that the solubility of sulfathiazole in tissue fluids is only about 150 mg. per cent.[8] This is, nevertheless, a ten to fifteen times greater concentration than can be obtained by oral administration. Moreover, the local use of sulfathiazole, or any other sulfonamide, in an abscess cavity or potential abscess cavity places the greatest possible concentration of the drug on the pus itself. Oral administration of the drug fails to do this. Woods[10] and MacLeod[5] showed that para-aminobenzoic acid, which is present in pus reduces the effectiveness of the sulfonamide. The inhibitory action of a sulfonamide results in interference with the bacterial enzyme reaction necessary for the utilization of para-aminobenzoic acid, a compound essential to bacterial metabolism. Therefore drainage of a mastoidectomy wound not only permits washing out of the sulfonamide, but also decreases the concentration of the bacterial inhibitory action of the drug.

The absorption rate of a sulfonamide used locally depends in a large measure upon the vascularity of the part. As the pathologic process of mastoiditis usually walls off the infection, the absorption is small and the local therapeutic effect is great. Consequently, there is no danger of too high blood concentration from local use of a sulfonamide in mastoidectomy wounds. Crile[2] indicated that the blood concentration after local use of sulfanilamide is approximately one-fifth that of equal amounts given orally. Guerry and Putney, and I, too, have been unable to detect sulfathiazole in the blood when the drug was used in mastoidectomy wounds.

Osgood[8] carried out a series of remarkable experimental studies concerning the relative bactericidal and bacteriostatic action of various sulfonamides. The principal steps of his research are as follows: He produced a living, human culture medium from marrow cells, which he obtained from healthy adults by means of sternal puncture. These marrow cells he grew in a preparation composed of 35 per cent human placental cord extract and 65 per cent balanced salt solution, which is essentially similar to human serum. To a known number of cells in a given amount of culture medium, he introduced a known number of bacteria and also added a known amount of either a sulfonamide or some other drug. Then by microscopic examination of smears made at varying intervals, determinations were made (1) of the approximate number of bacteria still present and (2) of the effect of the bacteria and the drug upon the cells of the human culture medium.

Among many interesting findings, Osgood observed that the most bacteriostatic sulfonamide for almost all types of bacteria, including hemolytic streptococcus, was sulfathiazole. He observed, also, that sulfanilamide was the least bacteriostatic, and that the bacteriostasis of sulfapyridine and of sulfadiazine was somewhere in between that of sulfathiazole and of sulfanilamide. Furthermore, he showed that the sulfanilamide concentration had to be six times greater than that of the sulfathiazole before an equal bacteriostatic effect could be obtained, and that in this high concentration sulfanilamide killed large numbers of human marrow cells. Hence he concluded that in practically every instance the drug of choice should be sulfathiazole.

CONCLUSIONS

From the data obtained, the following conclusions may be drawn:

1. The use of a sulfonamide in a snugly closed mastoidectomy wound, without drain, accelerates healing, decreases postoperative drainage, and lessens postoperative hospitalization.

2. Sulfathiazole is the drug of choice for use in mastoidectomy wounds.

SUMMARY

A study is presented of the local use of sulfathiazole in 84 unselected mastoidectomy wounds. Some of the wounds treated with sulfathiazole were closed tightly without drain, others were closed after a soft rubber drain had been inserted. The procedure followed in 52 simple mastoidectomies included in the present series is discussed in detail, and the results obtained in this group are compared with those obtained by two other investigations using sulfanilamide and sulfathiazole, respectively, in similar cases. It was found that the best results could be expected when the wound is closed tightly without drain after sulfathiazole implantation.

A brief resumé is also included of the research of various investigators relative to the local bactericidal and bacteriostatic action of sulfonamides and the effect of these drugs on wound healing.

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LXXXIX

THE EFFECT OF ANDROGEN THERAPY ON THE VOICE AND VOCAL CORDS OF ADULT WOMEN*

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During the course of administering androgens in the treatment of female endocrine disorders, the observation was made that a number of the women developed changes in the character of the voice and in the physical appearance of the larynx which could not be attributed to anything but the hormone therapy.[1-4] In view of the increasing use of male sex hormone in the treatment of endocrinopathic gynecologic disorders, it seemed important to study these laryngeal changes and determine whether they can be prevented.

The observations in this communication were made from a series of over 400 patients who were treated with various androgens; viz., testosterone propionate administered intramuscularly, methyl testosterone orally, testosterone by implantation of pellets, and testosterone in solution in propylene glycol sublingually, in the Gynecological Endocrine Clinic at The Mount Sinai Hospital during the past four years. The series comprises cases of dysmenorrhea, menometrorrhagia, premenstrual tension and menopause syndrome. The patients varied in age from 16 to 61 years. In this report we wish to describe our observations in 23 cases that manifested striking vocal and laryngeal changes as a result of the intramuscular administration of testosterone propionate.

The total amount of testosterone propionate which these patients received varied from 225 to 3,000 mg. over periods extending from one to 22 months, given in individual doses of 25 or 50 mg. two or three times weekly.

The effect of the administration of androgens upon the larynx was evaluated by vocal changes as noted by the patient or others and by physical alterations of the vocal cords as observed by indirect

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mirror laryngoscopy. The patients were interviewed and examined at intervals varying from two to six weeks during the period of androgen therapy, and were observed for periods varying from four months to almost three years following the discontinuation of therapy.

The vocal complaints were hoarseness, huskiness, voice with masculine characteristics, constricting or tightening feeling about the larynx. Thirteen of the patients presented the complaint of hoarseness or huskiness and the voice of some of these possessed a phonesthenic quality. In the remaining 10 patients there were varying degrees of lowering of the pitch. In 7 instances the pitch and quality of the voice were distinctly masculine in character. The voices of several of the patients progressed from the hoarse to the masculine type while under observation. Seven patients described a constricting or tightening sensation about the larynx. This was, as a rule, constantly present once it was manifested and became aggravated after much use of the voice. The voices of all these patients fatigued easily.

The vocal cords consistently exhibited a characteristic picture. They presented a pale grayish color, which became off-white as the process progressed, and they were swollen and enlarged. The impression obtained was that the enlargement was the result of a diffuse edema. Caudal extension of the edema in the depths of the vocal cords was often noted, particularly in the advanced cases. As a result, the subglottic surfaces of these vocal cords were visible on indirect laryngoscopy. Imperfect approximation of the posterior portions of the vocal cords was also seen in many of the cases. In addition, in most of these patients, injected vessels ran the length of the vocal cords. The appearance of the larynx resembled the picture seen in allergic conditions rather than that seen in infectious processes.

The degree of alterations of the vocal cords varied greatly in the cases observed. We have designated the extent of the swelling and injection from 1 to 4 plus. Ten showed 1 plus swelling, 7 showed 2 plus, and 5 showed 3 plus. In the one case graded 4 plus, the vocal cords swelled to about three times their normal size. The intensity of vocal symptoms usually ran parallel with the degree of changes of the vocal cords.

It is of considerable clinical significance that in some instances vocal disturbances as well as physical alterations of the vocal cords persisted after discontinuation of the androgen therapy. In other

cases a change in the voice continued after regression of the swelling and congestion of the vocal cords. The latter did not return to a completely normal state but acquired a thickened and pale appearance.

Persistence of vocal symptoms and physical changes of the vocal cords after discontinuation of the androgen therapy were observed in 11 patients. In 3 patients slight huskiness, lowering of the pitch and phonesthenia are present two and one-half years after discontinuation of the therapy. The vocal cords of these patients appeared moderately thickened. In each of these cases the total dosage exceeded 900 mg. of testosterone propionate. In a fourth patient, who had received 2,200 mg., coarseness of the voice and 3 plus changes in the vocal cords have persisted for twenty-eight months and in another (1,750 mg.) sixteen months after cessation of androgen therapy. The voices in 3 other patients have retained their masculine quality for a period of over a year, but the vocal cords of these women at the end of that time appeared normal. In 3 other patients there was still evidence of vocal change and vocal cord alterations five, six and ten months, respectively, after discontinuation of the androgen therapy. In the remaining 12, return of normal voice and complete regression of vocal cords to the normal state occurred from one to three months after stopping the androgen therapy.

DISCUSSION

Shortly after the institution of androgens in gynecologic therapy, it was noted by a number of observers[1-3, 5-12] that masculinization phenomena, such as hoarseness, hypertrichosis and enlargement of the clitoris, developed in some of the patients.

It is interesting in this connection to note that Leonard[13] reported that singing occurred in female canaries following the administration of testosterone propionate. It has also been observed that in human males with hypogonadism manifesting characteristic clinical signs and symptoms of androgen deficiency including high-pitched voices, testosterone propionate has produced deepening of the voice as well as other secondary male sex characteristics.[14, 15]

The vocal changes which occur at puberty are a part of the secondary sex characteristics which result from the sex hormone activity. Although the vocal range changes both in the male and female at puberty, the pronounced lowering of the range (*a* [103 double vibrations] to *e* [162 double vibrations]) in the male is most likely due to the great preponderance of androgens.

In the female, on the other hand, it is reasonable to suppose that the range (*a* [217 double vibrations] to e^1 [320 double vibrations]) assumes a higher position in the musical scale because of the dominance of the estrogenic influence.

The primary factors which determine the vocal pitch are the size of the larynx and vocal cords and the position of the larynx in the neck. At puberty the larynx of boys undergo rapid increase in size. The vocal cords become 1 cm. longer in boys whereas in girls the vocal cords increase in size from 3 to 4 mm. At the same time there is a descent of the larynx in its relation to the other structures of the neck. This occurs much more noticeably in boys. Anomalies in sexual maturation exercise a marked influence in the development of abnormal secondary vocal characteristics.

The observations which we have made in patients receiving androgen therapy correspond to the changes in the vocal cords of males at puberty as noted by Seth and Guthrie.[16] They observed that swelling and redness occur in the laryngeal mucosa in this period of life, which, they warn, may lead to the erroneous diagnosis of laryngitis. It was their impression that the first sign of a change of voice is slight huskiness due to imperfect closure of the posterior part of the glottis.

Thus, it appears that the vocal and laryngeal changes which occur in females who receive large doses of testosterone propionate are similar to those which develop in boys at puberty. In the majority of the women under our observation, discontinuation of the male hormone resulted in the return of the normal voice and in the complete regression of the physical alterations of the vocal cords. In the group of women who received the large doses of testosterone, impairment of the voice was present as long as two and one-half years after cessation of androgen therapy, although slight improvement had apparently occurred. The voice in these patients acquired a husky quality with the tendency to be phonesthenic and to fatigue after use. Although the swelling and congestion of the vocal cords disappeared, as a rule, in these cases, the vocal cords became thicker and more fibrous than those usually seen in women. It should be noted that the voices of three patients retained their masculine quality although the vocal cords had apparently returned to the normal state. This suggests that an alteration had occurred in the vocal mechanism. The constriction of the throat experienced by some patients may be due to the action of the androgens on the depressor muscles of the larynx which, it is believed, contributes to the descent of the larynx at puberty.

Studies of the biologic effects of testosterone propionate have shown that the endocrinological effects vary with the dosage administered. If more than 500 mg. is administered during the course of one menstrual cycle, the secretion of gonadotropic hormone by the pituitary is suppressed,[10, 17] ovulation is inhibited,[2, 18] the production of estrogens and progesterone by the ovary is stopped[2, 3, 10, 19] and the usual cyclical morphological changes in the endometrium fail to occur, the endometrium and vaginal mucosa revealing varying degrees of involution indicating estrogen deficiency.[4, 20-22]

It is noteworthy that of the 23 patients who developed laryngeal complications, all but two, who had received 225 and 250 mg. respectively, were given more than 500 mg. of testosterone propionate. This amount has been established as the threshold for the induction of masculinization (arrhenomimetic) phenomena.[2, 3, 10, 12, 22]

It appears that as soon as one exceeds this dosage level, the incidence of arrhenomimetic phenomena, including vocal symptoms, mounts rapidly. Fortunately, testosterone propionate exerts its therapeutic effect below this level. During the early stages of androgen experimentation, in an attempt to determine the range of the biologic and therapeutic effects of androgens, huge doses were administered (up to 3,000 mg.). Subsequent studies revealed that it was not necessary to administer doses which induce the suppressive and masculinizing effects to treat the average case of functional menometrorrhagia or dysmenorrhea. Approximately 200 mg. of testosterone propionate per month were found to be adequate.[3, 11, 12] With this dosage the risk of vocal and laryngeal changes is negligible, as is indicated by the fact that no vocal symptoms occurred in over 200 patients treated in this manner. Furthermore, in the two cases in which vocal changes did occur with doses of less than 500 mg., they were slight in degree and rapid restoration to the normal state occurred after the discontinuation of the androgens. As a further safeguard, we have made it a routine practice to control every patient receiving androgens with weekly vaginal smears. At the first indication of androgenic effect on the smears,[2, 3, 20, 22] therapy should be discontinued. It has been conclusively demonstrated that evidence of androgenic effect in the vaginal smears can be detected several weeks before the appearance of clinical manifestations of androgen overdosage.[2, 3, 20, 22] A simple technic of staining the smears[23, 24] which has been employed in our clinic for the past five years makes this valuable method of controlling androgen dosage available for office use.

SUMMARY

In a series of over 400 patients who received androgen therapy for various endocrinopathic gynecologic disorders, 23 developed vocal symptoms of hoarseness, huskiness and masculine pitch associated with structural alterations of the vocal cords from the intramuscular administration of testosterone propionate. The vocal cords in these cases presented, on indirect mirror laryngoscopy, a characteristic picture of generalized suffused swelling with injected blood vessels running the length of the cords. A pale off-white color with caudal extension of the swelling so that the subglottic surfaces of the cords could be seen were consistent features of the laryngeal picture.

The total dosage in all except two cases was over 500 mg. of testosterone propionate. Persistence of vocal disturbance and physical alterations of the vocal cords existed for varying periods of time up to two and one-half years after discontinuation of androgen therapy in 11 cases.

This study suggests that there is a striking similarity between the vocal and laryngeal changes in women who receive androgen therapy and the physiologic development of the voice and larynx in boys at puberty.

It is evident from these observations that in order to avoid the production of vocal and laryngeal changes in women receiving androgen therapy, the dose must be kept below the threshold for these phenomena. For the average woman, this threshold is approximately 500 mg. of testosterone propionate per month. The therapeutic dose of 200 mg. per month recommended makes it possible to utilize the valuable therapeutic properties of testosterone propionate without running the risk of causing laryngeal complications. As an additional safeguard against androgen overdosage in susceptible individuals, it is recommended that weekly vaginal smears be taken and that therapy be discontinued if the androgen effects appear in the smear.

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XC

PATHOLOGICAL EXTENSION TO THE TEMPOROMANDIBULAR FOSSA AND TO THE FACIAL CANAL FROM OTITIS MEDIA*

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The close proximity of the middle ear cavity to the temporomandibular fossa is not yet fully appreciated. The rapid advance of aviation with the consequent exposure of a large portion of the population to sudden changes in atmospheric pressure on the two sides of the drum membrane make obligatory the accumulation of exact data on the normal and pathological anatomy of this region. Costen and others have frequently observed and reported neuralgias, eustachian tube and ear symptoms due to disturbed function of the temporomandibular joint. Such cases arise from malocclusion of the jaws. They occur most frequently because of loss of the molar teeth.

That a pathological condition in these regions may be more or less reciprocal is not generally realized. Sporadic cases occur clinically in which an individual with an otitis media may develop a painful temporomandibular joint. Movement of the jaw may be difficult.

The modus operandi of such pathological states has been clarified by the study of serial sections of the region. During the course of routine collection of temporal bones, specimens from eight cases have been procured which were sufficiently extensive, lateralward, to include a view of the fossa in relation to the external auditory canal, middle ear and eustachian tube. The ages of these patients were 7, 27, 36, 42, 46, 53, 55 and 65 years respectively. Bilateral specimens were procured in two cases, giving a group of ten bones for microscopic study of this particular area. All bones were cut horizontally, except one pair which were sectioned vertically at right angles to the long axis of the petrosa. In this relatively small group unclosed sutures were found to extend bilaterally into the middle ear cavity in three cases. In one case the suture was 2 mm. in width

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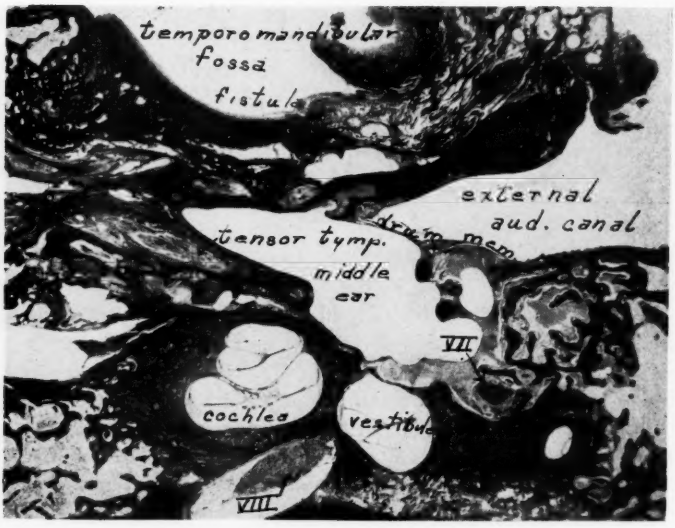

Fig. 1.—No. 8565 Rt. Horizontal section through the inner ear, middle ear, external auditory canal and a portion of the temporomandibular fossa in a 53-year-old white male with otitis media. Note the exposed position of the eighth nerve and the pus entering the canal.

and was filled with very delicate areolar tissue. In the others it was filled with dense fibrous tissue as one would expect. In some cases in which an open suture was found it extended only to the external auditory canal and would presumably offer little or no hazard. Some of the unclosed sutures opening into the middle ear occurred in relation to *iter chorda anterior*. In others it occurred more posteriorly, lying opposite the facial nerve or the distal portion of the tensor tympani muscle. The anatomic variation making possible this lesion appears to be due to the extent of development of the tympani bone.

Fig. 1 illustrates a case in this series (8565) from a white man, 53 years of age. He came into the hospital in a moribund state with meningitis of otitic origin due to pneumococcus type III. He expired within 18 hours after admission.

Serial sections of the right temporal bone block revealed that an extension of the infection had occurred laterally with a definite fis-

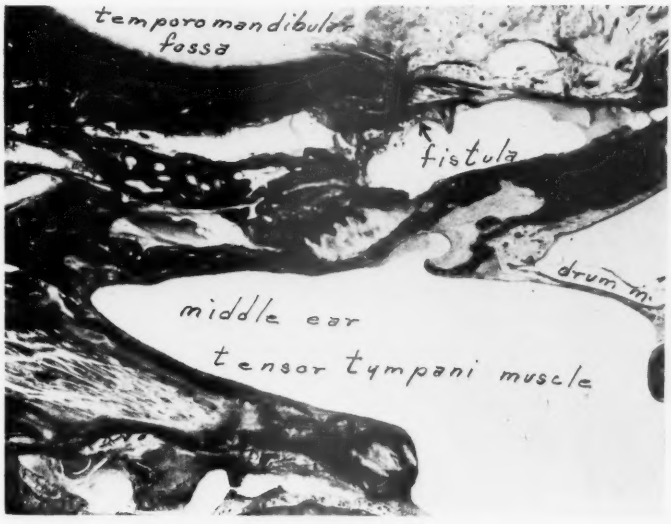

Fig. 2.—No. 8565 Rt. Detail of Fig. 1, to illustrate the fistula from the middle ear cavity into the temporomandibular fossa.

tula into the temporomandibular fossa as illustrated in detail in Fig. 2. This case also illustrates another anatomic variation. It may be readily seen in Fig. 1 that the bony facial canal is incomplete and the nerve is bathed in pus on the middle ear side. Such an anatomic condition is normal for the infant. The aperture in the facial canal posterior to the oval window marks the point of passage of the stapedial artery in the fetus as it makes the second aortic arch. The artery disappears in the third fetal month but the aperture remains throughout early infancy. In occasional cases, as in the one illustrated, it may persist in the adult. This aperture is of medico-legal significance to the surgeon. It is important for him to know whether or not a facial paralysis existed before any surgical procedure is instituted in the mastoid region. In the case of infants it is usually the mother, rather than the examining physician, who notes that the baby fails to laugh or cry on both sides of its face. In the case of the older child or adult it is usually some member of the family who has noted that the patient failed to wink one eye or to articulate properly in speech.

A careful check by Dr. Edward Lyman on the pre-hospital history of the case here illustrated failed to reveal a history of facial paralysis or of temporomandibular joint difficulty.

The author is indebted to M. Cook, M.D., for the photography of the sections.

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XCI

EXPERIMENTAL AND CLINICAL OBSERVATIONS REGARDING THE PHYSIOLOGY AND PHARMACOLOGY OF THE NOSE*

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The mucous membranes lining the nose and upper respiratory passages possess a high degree of sensitivity. Minute traces of such substances as chlorine, benzedrine, or ammonia in the respired air may lead to extensive reactions in a variety of structures or organs, both local and remote. The observations and experimental results described in this paper deal with the general character of some of these reactions, and with the basic mechanisms by which they are rendered possible. There are many problems yet to be solved in this general field. It is probable that, in some instances, enlightenment may be more readily gained through a study of the action of drugs on these respiratory passages than in any other way. Many drugs, hormones, and the like, which may be introduced into the circulation at points remote from the nose can exercise influences, which in some instances may be very profound, on these upper respiratory passages and their related structures. It is hoped that the approach to these problems which the following experiments illustrate may be helpful to others who work in this general field, either in the clinic or in the laboratory.

Figure 1 illustrates one of the most striking features of the physiology of the nose. In this record the upper tracing shows the air volume changes in the nose; the middle tracing shows the blood pressure as recorded by a mercury manometer connected with the right femoral artery; and the lower tracing shows the respiration as recorded by a tambour connected to a side tube of the tracheal cannula.

The animals (dogs) which we have used were usually anesthetized with ether, but we have also used other anesthetics. All drugs used in the experiments except ether were injected intravenously.

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The nose records (Fig. 1) were made by a very sensitive tambour which was connected by rubber tubing to both nares by means of a Y-tube and two short glass tubes with bulbed ends which were inserted (well vaselined) into the anterior nares. By means of a soft sponge rubber ball attached to the end of a metal rod, the soft palate was pressed upwards and backwards against the posterior wall of the pharynx (air tight). In this manner both of the nasal cavities and the adjacent connecting sinuses could be made to act as a closed chamber, the walls of which will vary in thickness with changes in the blood vessels, the lymphatics, and perhaps in other ways. A sensitive tambour was used to record the changes in the volume of air which these combined cavities contained from moment to moment. It should be added that the rubber ball on the end of the metal rod (nine inches long) was pushed back into the animal's mouth to a point just beyond the hard palate. The rod, with the ball on the soft palate, was then pressed backward (upward) by means of a clamp which passed through the mouth from side to side (over the rod) and then around the outside of the nose. By means of two wing nuts the clamp could be closed down to shut off the posterior opening behind the choanae. The front end of the rod rested against the middle upper incisor teeth, which thus served as a fulcrum for the rod, while the clamp pressed the ball firmly against the soft palate to push it back against the pharyngeal wall. This procedure represents a considerable improvement over an old method which was perhaps first used by Tschalussow[3] in 1913 and which has since been employed by a number of other investigators,[5-10] all of whom used some method of packing off the posterior opening of the nasopharynx.

The tubes which we have inserted into the anterior nares must be held fast in place. We have done this either by means of a special clamp,[1, 2] or, more easily, by tying a soft wire of iron, copper or aluminum, number 18, around the tip of the nose just in front of the position which the glass bulbs would occupy inside the nares.

We have recorded blood pressure from the femoral artery in order to avoid complications from the carotid sinus or the carotid body which would occur if the common carotid artery were used.

If tracings are made from an anesthetized dog under these circumstances, it is found that stimulation of an intact vagus nerve for a few seconds will produce results such as are illustrated in Fig. 1. This shows the prompt and vigorous constriction (down stroke, i.e., diminished air pressure in the nose record) which occurs in the nasal vessels. This change in the nasal walls may or may not be

independent of the general systemic blood pressure. It is to be noted that in Fig. 1 there is little change in the pressure in the femoral artery, for in this animal the inhibitory action of the vagus upon the heart is very slight. On the other hand, a marked fall in general blood pressure may produce striking changes in the nasal vessels, for a sudden fall of blood pressure is likely to produce a marked asphyxia and this causes a stimulation of the central sympathetic mechanism, which may then indirectly affect the nasal vessels. Stoppage of the respiration from stimulation of the vagus may also lead to asphyxia and thus affect the nasal arterioles. A marked sudden fall in systemic blood pressure, if accompanied by changes in the arterioles and capillaries of the nasal and sinus walls, may also greatly affect the volume of air which these chambers will contain at ordinary atmospheric pressures. Greatly reduced atmospheric pressures, as those encountered in high altitude flying, might also effect changes in the nasal and the aural circulation; but, in general, these would probably be entirely overshadowed by more profound systemic phenomena.

Figure 1 shows well the marked influence which the sympathetic vasoconstrictor nerves exercise on the nasal vessels. Obviously these fibers pass to the nose through the cervical sympathetic nerves, which in most animals run in the same sheath with the vagus fibers and those of the aortic nerve (formerly called the depressor nerve). Vasodilator fibers for the nose apparently do not exist in the cervical sympathetic trunks. Tschalussow[3, 4] believed he had demonstrated the presence of vasodilators for the nasal and the sinus regions in the vidian nerve. Sternberg[8, 9] believed his experiments corroborated this finding. Other later writers have accepted their conclusions. So far we have not repeated Tschalussow's experiments on the vidian nerve, but the tracings which he and Sternberg present in this connection seem convincing. None of these authors mention any effect which the secretory nerves to the mucous glands of the nose might have on volume changes. In this connection it might be recalled that the secretory nerves to the lachrymal glands pass outward in the seventh cranial nerve but have their ganglionic way stations in the sphenopalatine ganglion, while cranial secretory and vasodilator fibers for the salivary glands run together in the fifth, seventh and ninth nerves and have their ganglionic way stations in the otic, the submaxillary, etc., ganglia. Electrical stimulation of these nerves leads to both secretion and vasodilation in the involved glands. True sympathetic secretory nerves to such glands are usually accompanied by vasoconstrictor fibers which would be affected by electrical stimulation of the mixed nerve.

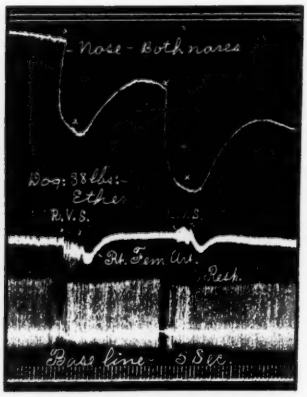


Fig. 1.—Tracing showing the intranasal air volume, blood pressure (femoral artery) and respiration. *R.V.S.*, right intact vagus stimulated. *L.V.S.*, left intact vagus stimulated.

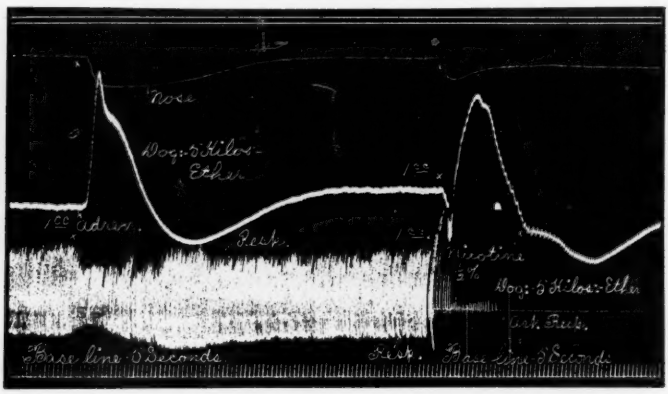


Fig. 2.—Tracing showing the action of adrenalin and of nicotine on the intranasal air volume, the blood pressure and the respiration.

Figure 2 illustrates two different types of action of drugs on the nasal vessels, viz: that of adrenalin and of nicotine. Adrenalin stimulates the endings of the vasoconstrictor nerves, while nicotine stimulates the sympathetic ganglia on the course of these fibers. In addition to the blood pressure and respiratory changes shown, there is produced a constriction of the nasal vessels. This figure also illustrates another feature which we believe would hold good in humans also. This animal was a small, young dog. The nasal records are relatively small and we believe this is largely dependent on the underdevelopment of the nasal and associated structures such as those of a small child. There is also to be considered in this experiment the double effects of both vasoconstrictor and vasodilator stimulation. For adrenalin stimulates the endings (adrenergic action) of both these sets of nerves, and the ultimate result depends on the preponderance of constrictors over dilators. With nicotine a corresponding action originates in the ganglia.

Sternberg and others concluded that the vidian nerve carried mainly vasodilator fibers, but that a much smaller number of vasoconstrictor fibers were also present in the mixed nerve. The ganglionic action of nicotine and its congeners would, of course, affect both sets of fibers.

Figure 3 illustrates an especially interesting phenomenon. In this experiment one-half cc. of adrenalin (1:1,000) was injected into the femoral vein. A prompt and very marked shrinkage of the spleen (oncometer tracing) and of the nasal vessels occurred. Thus the visceral organs react in the same way and in a corresponding degree as do the walls of the nasal passages. The extensive experiments of Mudd, Grant and Goldman,[11-15] showing the shrinkage of the nasal vessels by reflex action when the body is chilled, at once recall the conception, formerly much emphasized, that exposure to cold or drafts might lead to the development of nephritis. It is interesting to note how promptly the nasal vessels respond to these injections of drugs when they are made into vessels far removed from the nose. It seems obvious that under special conditions enough adrenalin may frequently be discharged from the adrenal glands into the blood stream to produce marked changes in the nasal walls. And this, perhaps, also holds true for some other chemical substances when they are formed in the body. But it is interesting to recall that a fair number of cancer patients have been "packed in ice" for five days without "catching cold." Ordinarily, however, chilling of the body surface leads to shivering, and this perhaps to

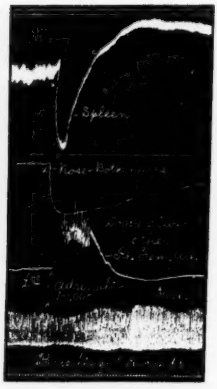


Fig. 3.—Tracing showing the relative action of adrenalin on spleen volume, nasal volume, blood pressure and respiration.

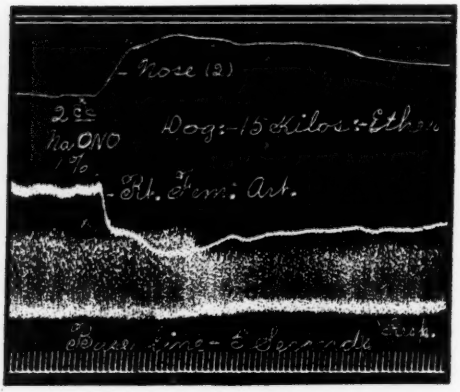


Fig. 4.—Tracing showing the action of nitrites on nasal volume, blood pressure and respiration.

stimulation of the adrenal glands, while at the same time there may be an increase in the secretion of mucus in the nasal passages.

A number of drugs which are in common use may cause quite extensive changes in the nasal passages. It seems obvious that in most cases these changes can be demonstrated more easily and clearly by the method we have used in these experiments than in any other way. A method which has been used by a number of investigators[7, 16] consists in measuring variations in the resistance to the passage of air currents through the nose before and after the local application of various drugs. We present in the following pages a number of records showing both vasoconstriction and vasodilatation as recorded by our method. It should be kept in mind in examining all these records that the drugs are injected into a femoral vein and are not painted or sprayed locally into the nose. Yet the results in the nose are apparently exactly the same, except more accurately and effectively produced than would have been the case if the drugs had been applied locally. There is, however, one difference. After intravenous injection the effects in the nose come on immediately and they tend to disappear as soon as the blood stream can carry away the drug, either to destroy it or to eliminate it from the body, or to distribute it into other less active and more extensive tissues. But not infrequently rather lasting changes may be produced in the nose by drugs administered intravenously, even though other changes produced by the drug in the blood pressure, respiration, etc., may have rapidly disappeared. The mechanisms by which some of these variations are produced may not always be obvious. Many reflexes and obscure readjustments in the circulation, lymph flow, respiration, etc., must be present. Undoubtedly these same mechanisms play a part in the clinical course of many patients.

Figure 4 shows the action of sodium nitrite on the nasal walls. This is probably due to a direct depressant action on the muscle walls of the arterioles and the venules. But depression of smooth muscle fibers in the general structure of the walls of the nasal passages, and changes in lymph formation and lymph flow may also be involved. It is to be noted that in this experiment the nasal changes correspond exactly with those of the blood pressure, both as to extent and duration.

Figure 5 shows the nasal changes produced by ether. Undoubtedly changes similar to these shown here occur in many, if not all, patients who are anesthetized with ether for ordinary operations. It is to be noted that the nose changes may here not coincide with those of either the blood pressure or the respiration, for the effects

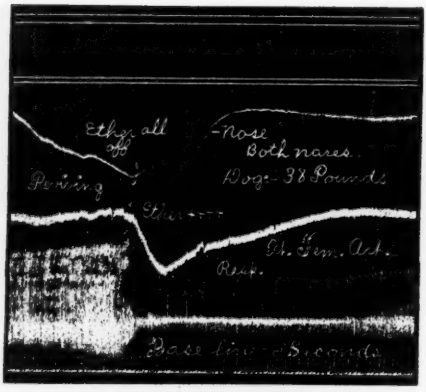


Fig. 5.—Tracing showing the action of ether on nasal volume, blood pressure and respiration.

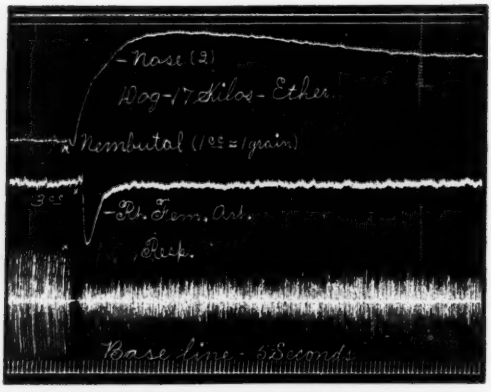


Fig. 6.—Tracing showing action of nembutal on nasal volume, blood pressure and respiration. Note prolonged nasal vascular dilatation, while the blood pressure returns almost to normal.

of ether appear to be mainly central and due to depression of the vasomotor centers in the medulla. However, an increase or a decrease of the respiratory movements may produce marked changes in the nose, apparently largely because of the shifting of a considerable quantity of the blood volume in the chest, the large veins, etc. This may best be seen in the case of violent convulsions. In Fig. 5 a marked nasal constriction (shrinkage of the mucosal walls) is seen at the beginning of the tracing labeled "reviving." Following this, the administration of concentrated ether vapor (Ether ++++) leads to a marked and persistent dilatation (upstroke) of the nasal vessels by depression of the vasomotor center.

Figure 6 shows a very similar effect produced by an intravenous injection of nembutal in an animal already lightly anesthetized with ether. In this case the dilatation (upstroke) of the nasal vessels seems to be almost specific, for the blood pressure, after a brief initial fall, returns almost to normal. The respiration is decreased by the nembutal, and one might expect this to lead to a certain degree of asphyxia with some stimulation of the medullary vasoconstrictor center and shrinkage of the nasal membranes. But the drug seems to counteract centrally any effects in this direction. This tracing must call to mind the present widespread and increasing use of barbiturates for intravenous anesthesia. One might well expect this marked dilatation of the nasal vessels and this swelling of the walls to lead to some nasal obstruction, snoring, etc.

Figure 7 shows the action of histamine on the nasal walls. It is to be noted that histamine, when injected rapidly into a vein, causes a marked fall in the general systemic blood pressure. The very great dilatation of the nasal vessels shown here is apparently due in a considerable degree to this general action. But it seems quite likely that histamine possesses some fairly specific action on the nasal vessels. The possibility that this drug, or some nearly related substance, may be connected with the symptoms produced by "colds," nasal anaphylactic manifestations, asthma, edemas, etc., has been suspected by many investigators. Interesting observations bearing on this general subject have been published by Buhrmester and Wenner.[17] The recent experiments of Katz and Cohen,[18] in which they found that blood cells from sensitized animals release part of their histamine into the plasma when brought into contact in vitro with the antigen and their suggestion that, at points where the tissues may come into contact with the allergens to which the patient is sensitive, such as the digestive or respiratory tracts, enough histamine may be released from the blood cells circulating through these areas to produce local reac-

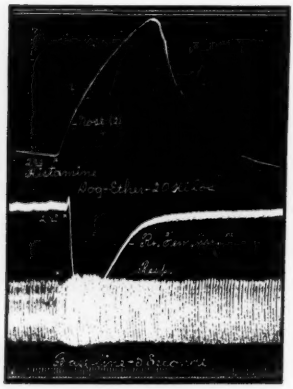


Fig. 7.—Tracing showing the action of histamine on the nasal vessels, blood pressure and respiration.

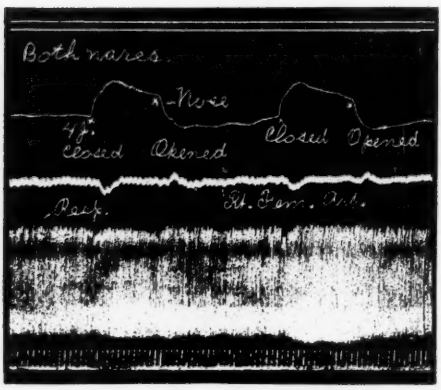


Fig. 8.—Tracing showing the changes produced in nasal air volume are shown. Both internal and both external jugular veins were clamped off and, after an interval, reopened. Repeated a second time.

tions, seem signficant. Further evidence in this direction is given by the latest experiments of Katz[19] in which he finds that, if, in a sensitized patient, the surface of the skin be denuded by cantharides blisters, then application of Locke's solution to the raw surface will extract histamine from the skin if the antigen be injected hypodermically into the area, but not if the antigen be omitted. One can scarcely refrain from speculating on the possibility that, through some round-about processes as these, bacteria may find conditions on the nasal membranes suitable for lodgement and growth with all the ensuing phenomena of acute inflammation and, probably, with accompanying general systemic symptoms. The profound effects produced by histamine on the nasal membranous walls would seem to indicate that this substance plays some part in acute inflammatory processes in these areas.

Figures 8 and 9 should be considered together. In the first of these, both external and both internal jugulars were clamped simultaneously in the neck. The result is a prompt congestion of the nasal walls and the tambour record rises. The congestion, however, is not so great here as one might be inclined to suspect. In patients, the wearing of a tight collar may tend to produce this same type of result. In Fig. 9 the right and left innominate veins were clamped in the chest. This blocked nearly all venous drainage from the head and also the subclavian veins from the forelegs. One might expect this greater venous blocking to cause more congestion in the nose, but it seems that, under these experimental conditions, the large veins of the forelegs and other adjacent areas act somewhat like a buffer reservoir which accommodates a larger volume of the blocked off venous blood. We have been especially interested in these blocking-off results because of their clinical significance. Here one must bear in mind the difference between dogs, with the body nearly always in the horizontal position, and man in whom provision has been made for maintaining an efficient circulation in either the horizontal or the upright position. Perhaps the most common clinical phenomenon depending on these mechanisms is the congestion and nasal obstruction experienced by many patients on lying down. The processes by which this obstruction is brought about are by no means always obvious. In general, if such a patient lies for a while on the left side, then that side of the nose becomes obstructed while the right (upper) side remains open. But if the patient turns over and lies on the right side, then, after an interval, the right side closes off while the left rather slowly opens. Probably in some degree this phenomenon occurs in all persons, normal as well as abnormal. But if the process becomes marked, the swelling in the nose may become uncomfortable

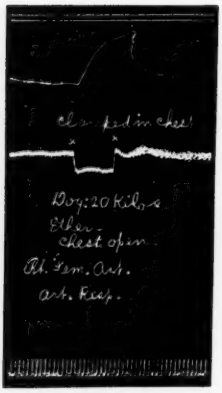


Fig. 9.—Tracing showing changes in nasal air volume. Both innominate veins were clamped in the chest. The nasal volume promptly increased. After an interval the veins were reopened and the nasal volume returned to normal.

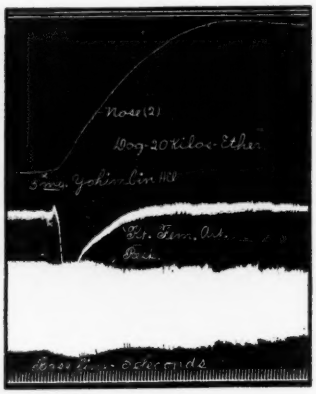


Fig. 10.—Tracing showing the action of yohimbine on nasal volume, blood pressure and respiration.

or painful and the patient will be forced to sleep most of the time in such position as to keep the disturbing nasal congestion at a minimum. From experience we have been led to wonder what effect this postural requirement may have on other organs or systems of the body. For example, will the stomach be affected by a prolonged lodgement of strongly acid gastric juice in the fundus or in the pyloric end of the stomach, and can this have a bearing on gastric disturbances or ulcer formations? How will the heart and the great blood vessels in the chest and abdomen be affected? It seems that here confusion may arise as to which is cause and which effect. One of us has seen a patient, a woman about sixty years old, in whom a condition exactly the reverse of that noted above was present. That is, in her case, when she lay down on one side, the lower side of her nose opened up, while simultaneously the upper side became congested and closed off. If she turned over and lay on the reverse side, then the lower side again opened up and the upper side became closed. By what mechanism could this be brought about? Obviously the vasomotor nervous control of the blood vessels in the nasal passages could effect these changes, but it seems very unlikely that this is the chief cause of such alterations. Although some intimation has been given in the literature that stimulation of the cervical sympathetic on one side in the neck causes vasoconstriction in both sides of the nose, this does not explain congestion on one side and constriction on the other. At first thought, one would almost certainly conclude that congestion (venous?) in the lower side of the nose is due to gravity. But how could this explain the case noted above in which the lower side of the nose opened up while the upper side became closed off? We have suspected that a number of factors are involved in bringing about these phenomena. Fig. 9 shows that venous obstruction within the chest can readily affect the nasal vessels. And we have suspected that much of this type of swelling in the nose is due to conditions in the chest. Increased pressure in the innominate veins (as in Fig. 9) may be reflected up into the nasal vessels. The weight of the heart and the overlying lungs, as the patient lies on the one side or the other, may be sufficient in some individuals to affect one or the other of the innominate, or even the jugular, veins and thus indirectly affect the nose. Enlarged lymphatic glands or old adhesions in the upper mediastinum might also lead to a partial obstruction of the large veins in the chest. Such a mechanism as this might explain the case mentioned above.

We have also considered the possibility that a blocking off of the lymph ducts which drain the nasal walls might cause a nasal obstruction on one side. In dogs one may cannulate these ducts and

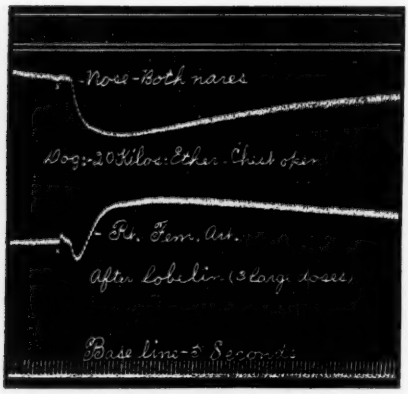


Fig. 11.—Tracing showing the action of octin (after lobelin) on nasal volume, blood pressure and respiration.

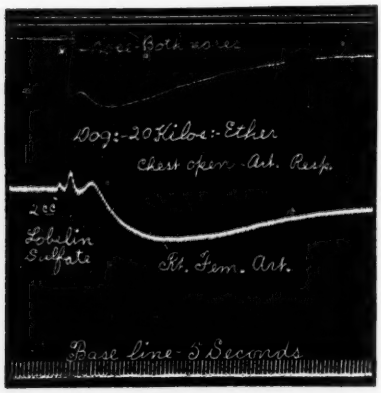


Fig. 12.—Tracing showing the action of lobelin (large dose) on nasal volume, blood pressure and respiration. This is a ganglionic action representing a mixture of stimulation and depression.

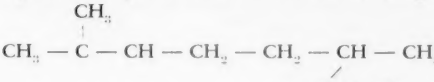
collect the flow. The pressure under which this lymph flows is very low, and a slight increase in pressure in the veins into which the ducts empty might easily retard the emptying of the ducts and thus lead to the blocking off of lymph drainage. When a patient of this type lies down on one side, there is usually an interval of from two to five minutes before complete obstruction of the lower side of the nose occurs. It seems that this interval may be ample in some cases for a sufficient amount of lymph to be held back and thus cause obstruction in the nose, if the duct is blocked off. An inflammatory swelling in the tissues or along the course of the lymphatics would, of course, here create a kind of vicious circle.

In man the internal jugular vein is larger than is the external. The reverse of this occurs in dogs. A partial obstruction of either of these veins, e.g., by pressure from the sternocleidomastoid, omohyoid or other muscle, or by other structures in the neck or chest, might be expected to affect the nasal venous drainage. And perhaps abnormal pressures in the auricles of the heart may also be reflected up to the nasal veins, for the valves in the larger veins here concerned are often incompetent, if present at all.

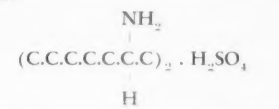
An interesting point concerning the pharmacology of the nasal vessels is brought to mind by Fig. 10 in which a very marked vasodilatation is produced by yohimbine.* This drug has a similar action on the erectile tissue in the reproductive organs. The similarity in structure and physiology of the cavernous tissue in these two regions has long been recognized.

Figure 11 shows the action of octin* on the nasal vessels. Here the drug produces marked constriction of the nasal vessels after the animal has received three large doses of lobelin by vein. The lobelin paralyzes the ganglia on the course of the vasoconstrictor nerves to

*Octin belongs to the aliphatic series and has the following formula:



Compare also an article by Arthur W. Proetz: "2-Amino-Heptane Sulfate as a Nasal Vasoconstrictor," The Annals of Otology, Rhinology, and Laryngology, 51:112-116 (March), 1942. This drug also belongs to the aliphatic series and has the following formula:



This drug is apparently a more active vasoconstrictor than is octin.

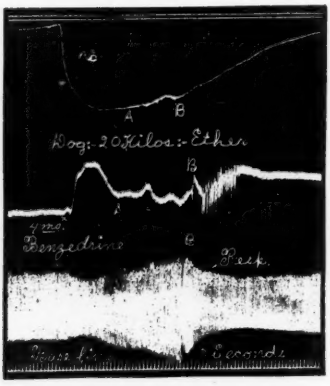


Fig. 13.—Tracing showing the action of benzedrine.

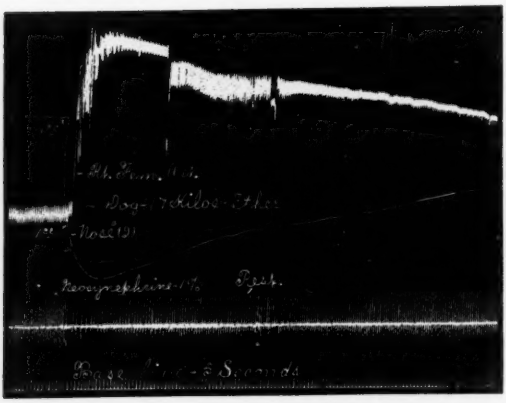


Fig. 14.—Tracing showing the action of neosynephrine.

the arterioles, so that octin stimulates the myoneural junctions of these nerves (adrenergic action). In other places, such as the ureters, the stomach, etc., octin apparently may cause relaxation of smooth muscle by stimulation of the endings of the inhibitory (true) sympathetic nerves.

Nicotine, lobelin and their congeners have in general two actions on autonomic ganglia. First, with small doses, they stimulate the ganglionic cells and may thus cause constriction of most of the arterioles of the body. This type of action on the nasal vessels is well shown in Fig. 12. Larger or repeated doses of these drugs paralyze the ganglionic cells and drugs can then affect the nervous mechanism only by acting beyond the ganglia, presumably at the myoneural junctions in the muscles or glands in which the nerves end. In the clinical literature one seldom finds mention of these ganglia, yet they are just as important for the autonomic system as are the anterior horn cells for the cerebrospinal nerves. These autonomic ganglia are motor in function, not sensory. In cases in which sensory (cerebrospinal system—medulla or cord) fibers run in connection with the autonomic ganglia it may be difficult to separate one set of fibers, or one function, from another.

The action of benzedrine on the nasal vessels is shown in Fig. 13. The corresponding actions on the blood pressure and respiration are also illustrated. A further (stimulant) action of the drug on the central nervous system is also seen beginning at *A*, at which point the animal starts to revive from the effects of the ether, and ending at *B* when stronger vapors of the anesthetic are administered.

The action of neosynephrine is shown in Fig. 14. A large dose was given and the effects are very pronounced. Aside from the nasal vascular constriction, the most striking feature of this tracing is the marked slowing of the heartbeats and the sudden drops in blood pressure when this has risen to a high level. This marked slowing (increased pulse pressure and missed beats) is mainly due to reflex inhibition of the heart as a result of the increased pressure within, and the mechanical stretching of the walls of the carotid sinuses. Blood pressure is taken from the femoral artery so that the carotid mechanisms may not be disturbed. It should be noted that some anesthetics especially depress these carotid reflexes.

A number of synthetic drugs having a sympathomimetic action are now on the market and more new ones are appearing from time to time. The action on the nasal vessels of two of these, paredrinol and propadrine, are shown in Figs. 15 and 16. It will be noted in this

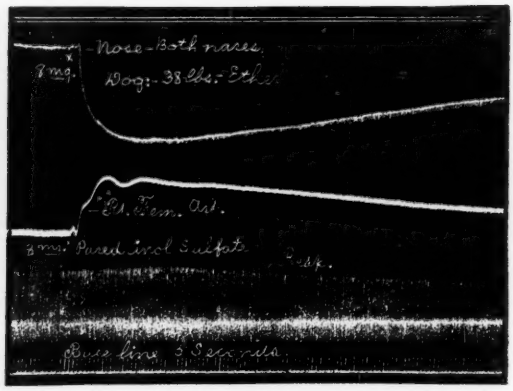


Fig. 15.—Tracing showing the action of paredrinol.

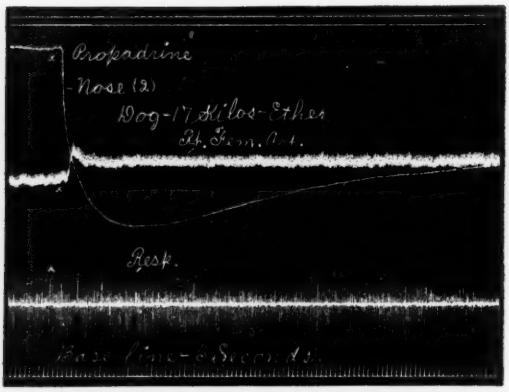


Fig. 16.—Tracing showing the action of propadrine.

series of tracings that in some cases the nose tracings seem exceedingly pronounced in comparison with the rise in blood pressure. This is often due to technical adjustments, magnification by the recording tambour, etc. But it was noted many years ago by Dale that sympathomimetic drugs vary a good deal in their relative activity on the various sympathetic nerve endings throughout the body, in the heart, kidney, intestines, bladder, ducts, etc. Perhaps this holds true for the nasal vessels also. Some of these compounds produce, when applied locally, a more pronounced secondary congestion than do others. The reason for this is generally obscure, but some of them perhaps stimulate the vasodilator nerve endings longer or more effectively than do others. A very small dose of adrenalin has been claimed to cause a fall in normal blood pressure in some animals, but we have not observed this in our experiments.

We should like to refer briefly to a clinical phenomenon which was well recognized by the laity in certain rural districts in this country some 50 to 75 years ago. This was long before any modern forms of sinus treatment[20] were introduced, for sympathomimetic drugs were unknown before 1895. This trouble was spoken of as "sun pain" and apparently consisted of a subacute or chronic involvement of the frontal sinuses (one or both). Perhaps in some instances other sinuses may have been involved, but typical effects apparently came from the frontal region. These pains were diurnal in character. From a condition of complete comfort in the early morning the patient began slowly to feel a dull aching pain in the frontal region. This pain began about eight o'clock and gradually grew worse and worse, as the sun rose, to reach a maximum about one o'clock. From then on the pain slowly decreased, as the sun descended, until about five or six o'clock when the patient again became entirely comfortable and could go on about his usual work. But the next morning saw a complete rehearsal of what had occurred the day before. This process was repeated every day, usually for a period of one to two weeks, after which the pain each day gradually grew less and less and finally disappeared. The onset also was usually of this gradual character. We have not found any mention of "sun pain," in this sense, in the literature and, as one old lady has stated, the disease seems to have "gone out of style." The close association of the pain with the time of the rising, the high ascent and the setting of the sun apparently gave rise to the name "sun pain." One of us has seen a patient of this type who had his pain at night, but not in the daytime. He was a night watchman. It would appear that at present little emphasis is placed on sinus infections or involvements of this type, but the diurnal character of these "sun pains" has been of special interest to

us. By what mechanism can this daily rehearsal be carried out? Its diurnal repetition resembles somewhat a daily infectious fever. It seems to be due to an increase in pressure in membranes which are already somewhat (but not painfully) swollen, as may be their state during the night. The suggestion has been made that the pain results from the occlusion of the ostium of the sinus and a slow filling up of the cavity by pus or secretion with a later slow opening of the ostium. We are of the opinion that this is not the correct, or at least not the complete, explanation. The pain is caused by processes occurring within the structure of the membranous walls of the sinus. Allergic phenomena would hardly explain involvement of one sinus only. In a single instance in which a patient was suffering from a "sun pain," we administered an ampule of amyl nitrite in the expectation that the general fall in blood pressure would lower the tension in the sinus region and possibly bring relief. But on the contrary, the drug made the pain worse. We did not know at that time that nitrites would actually increase the swelling and congestion within these membranes (Fig. 4).

We believe that this diurnal repetition of the pain is due to some deep-seated systemic cause. It may be related to some diurnal changes or processes in the central nervous system, such, for example, as those affecting the knee-kick or other more extensive reflexes. These processes may affect the pressure in the great veins which drain the sinus regions, or pressure in the auricles may be affected. And we have repeatedly tried to get some insight into what effects, if any, blocking of the lymph flow from the sinus regions may have in these cases. Aside from the use of aspirin, morphine, etc., to relieve the pain, it is difficult to apply drugs in such a way as to throw much light on the nature and etiology of these "sun pains." But since nitrites make them worse one might suspect that slow, careful, intravenous injection of a small dose of adrenalin might give some relief. But there are some elements of danger in this. However, among the considerable number of sympathomimetic drugs now available, there might be one which could be used intravenously and which might give relief as quickly as a nitrite relieves the pain of angina pectoris. But we would still like to know more about the physiology[21] involved in cases of this type.

Mosher[22] has called especial attention to the intimate relation of the veins of the frontal sinus membranous walls to those of the diploe of the frontal bone and of the scalp and the blood sinuses of the skull. These diploetic and associated veins are relatively large in size and of great importance in the spread of infection in these areas. The

marked changes which drugs produce in the vessels of the sinus walls we might expect to be carried over into these other related veins.

SUMMARY

By means of a special method which we have described we have studied the action of certain drugs as they affect the membranous walls of the nasal cavity and related structures. These drugs cover a wide range, both in chemical constitution and in pharmacological action.

We have discussed certain clinical phenomena in relation to the actions of the drugs employed and to the physiology involved in our experiments.

Dr. Samuel Iglauer has rendered extremely valuable assistance in various ways throughout the course of this work.

UNIVERSITY OF CINCINNATI COLLEGE OF MEDICINE.

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XCII

EFFECT OF EXPERIMENTALLY ALTERED AIR PRESSURE IN THE MIDDLE EAR ON HEARING ACUITY IN MAN*†

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BALTIMORE

Abnormal air-pressure in the middle ear affects the hearing acuity of many patients. Its influence can, however, seldom be determined because usually several other factors, such as mucosal edema, hyperplasia, fibrosis, adhesions, extravasated fluid, pus, etc., also contribute to the total impairment of the patient. To evaluate the possible effect of the factor of altered pressure, experimental methods are therefore necessary. The experiments to be reported were made on six subjects who have records of hearing acuity for several years past and who were free of infections of the upper respiratory tract or inflammation of the middle ear. That the changes in hearing were caused by the pressure changes created is proved by the fact that hearing acuity always returned to the original level after each experiment.

At each test thresholds were determined, with a Western Electric Co. 1-A audiometer in a sound proof room, for the following frequencies: 32, 64, 128, 512, 1024, 2048, 2896, 4096, 5793, 8192, 10321, 13004, and 16384 cycles per second. The importance of using this wide range of frequencies will become apparent later.

Pressure changes in the middle ear were produced by Valsalva's procedure. This method was employed instead of Politzer's method, tubal catheterization or air pressure apparatus in order to be absolutely sure not to exceed physiologic limits and to avoid any possibility of mechanical irritation of the eustachian tube or the middle ear. The pressures obtained are, therefore, within the range that occurs spontaneously in patients. Valsalva's procedure was carried out in the usual way. To increase the pressure in the middle ear the subject was asked to close the mouth and both nostrils and to try to exhale forcefully, and to decrease the pressure he was instructed

*From the Otological Research Laboratory, Johns Hopkins University.
†Aided by a grant from the John and Mary R. Markle Foundation.

to try to inhale forcefully. Sometimes the subject was asked to maintain the nasopharyngeal pressure, either positive or negative, while thresholds were determined with the audiometer. These tests are designated as: "during positive" or "during negative" Valsalva, respectively. On other occasions the nasopharnygeal pressure was released before thresholds were determined; these tests are designated: "after positive" or "after negative" Valsalva.

The pressure in the middle ear cannot be measured exactly without using procedures unsuited for the present experiments, because they change the normal functioning of the ear. Therefore, it was necessary to be satisfied with the approximate values obtained by using manometers in the external canal and in one nostril. These manometers record only the pressure in the external canal and in the nasopharynx, respectively, and are affected by other factors, such as movements of the jaw or soft palate. It is realized that in the positive Valsalva procedure the pressure in the nasopharynx is usually higher than the final pressure in the middle ear. The nasal manometer shows only the maximum pressure that may have been created in the middle ear. After releasing positive pressure in the nasopharynx the manometric readings for the external canal always decrease. This shift in manometric readings, however, gives some indication of what proportion of the excess air escapes through the eustachian tube. By combining the information from the two manometers, it has been possible to estimate approximately the final pressure in the middle ear to a degree that has proven satisfactory in correlating the changes in hearing acuity. On this basis the pressure changes have been grouped as slight or moderate (approx. 5 to 20 mm. Hg.) and as marked (approx. 20 to 40 mm. Hg.).

Sometimes in the positive Valsalva procedure the pressure dropped even below the original level, and in these cases otoscopic examination showed that the tympanic membrane had snapped back after release of the pressure in the nasopharynx. It is difficult to explain the creation of a negative pressure by this procedure; perhaps it is caused by an excessive contraction of the tensor tympani muscle. Permeability of the eustachian tube is undoubtedly a factor but deliberate attempts to repeat the phenomenon usually failed.

After the negative Valsalva procedure the pressure in the middle ear, as indicated by the manometer in the external canal, usually remained at a practically constant level until the next swallowing movement was made. If the negative pressure created was of marked degree (20 to 40 mm. Hg.), even the act of swallowing sometimes did not cause the eustachian tube to open. Individual

differences were noted, but all the subjects who participated in the experiments found it easier to maintain a negative pressure in the middle ear during the tests of thresholds of hearing than to maintain a positive pressure.

OBSERVATIONS

A slight positive pressure in the middle ear improved the acuity of hearing for the highest tones (Fig. 1, *A*, *B*). A moderate increase in the pressure caused an impairment of thresholds for low and middle tones (Fig. 1, *C*). With a marked degree of positive middle-ear pressure the thresholds for all frequencies below 2048 cycles per second became impaired by from 20 to 30 decibels (Fig. 1, *D;* Fig. 4, *A*, *B*, *D*). As the pressure increased, part of the improvement in hearing acuity for the highest frequencies usually was lost (Fig. 1). The thresholds for 4096 and 5793, in most experiments, showed the least change; sometimes the thresholds for these frequencies did not change at all. The impairment for 2048 and 2896 was seldom more than 10 to 15 decibels, even with marked positive pressure (Fig. 1, *C, D;* Fig. 4, *A*, *B*). Occasionally a much greater impairment developed for 5793 or 8192 than for neighboring frequencies (Fig. 4, *D*). Such "dips" occurred more often for 8192 than for 5793; they could not be deliberately produced in any of the observers. Rarely an impairment of thresholds for all frequencies, including the very highest ones, occurred; in these cases the impairment for the frequencies above 8192 was but slight compared to that caused by negative pressure in the middle ear (Fig. 3, *D*).

In tests made under the condition termed "during positive Valsalva" (see above), gradual release of the pressure resulted in a recovery of thresholds that started with low and middle frequencies; the last tones for which hearing acuity returned to the original levels were the highest ones. With sudden release of the nasopharyngeal pressure an immediate return to original levels occurred for the thresholds of all frequencies except the very highest. These sometimes continued to show slight improvement until after swallowing one or more times. In these cases a slight positive pressure no doubt was still present in the middle ear.

The effects on hearing acuity of negative pressure in the middle ear definitely differ from those of positive pressure only for the high tones. A slight negative pressure caused an impairment of thresholds for frequencies above 5793 cycles per second, in contrast to the improvement caused by a slight negative pressure (Fig. 2, *A*, *B;*

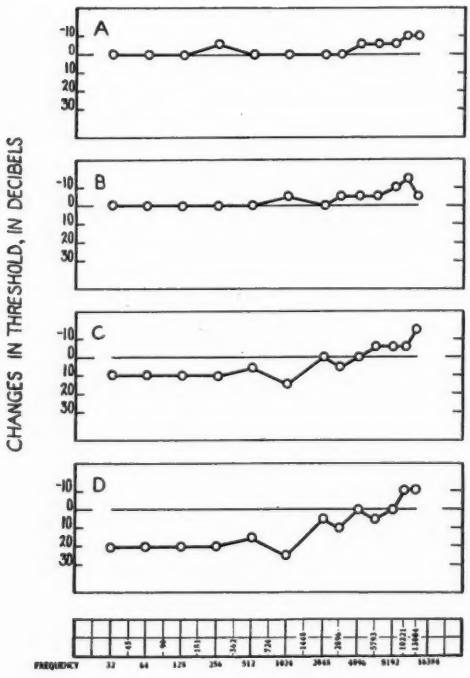

Fig. 1.—Changes in thresholds of hearing with different degrees of positive pressure in the middle ear. For convenience of reference the original audiogram is plotted as a straight line (zero) in each chart of this and of Figs. 2, 3, and 4.

Fig. 3, *A*, *B*). As the degree of negative pressure was increased, the thresholds for middle tones, and later for low tones also, became impaired (Fig. 2, *C*). With a marked degree of negative pressure, there was usually an impairment for all frequencies below 2048 of from 20 to 30 decibels, very similar to that caused by a marked degree of positive pressure (Fig. 2, *D*; Fig. 1, *D*). The impairment of hearing for the highest tones became greater as negative pressure increased; sometimes the highest frequencies originally heard by an observer could no longer be perceived at all (Fig. 2, *C*, *D*). As with positive pressure in the middle ear, negative pressure caused less impairment of hearing acuity for 2048 and 2896 than for lower frequencies. The frequency for which the hearing is least affected by negative pressure is 4096; sometimes the threshold for it even showed a slight improvement (Fig. 4, *C*). Markedly greater impairment than for neighboring tones was occasionally observed for the frequencies of 2896, 8192 or 10321 (Fig. 4, *C*). As with positive pressure, deliberate attempts to produce such "dips" were not successful.

The "during negative" and the "after negative" thresholds were, in most of the experiments, practically identical when negative pressure was suddenly released (Fig. 4, *C*). Recovery of thresholds to their original levels, during gradual decrease of negative middle-ear pressure, occurred first for low tones, later for the middle tones and finally for the very high tones. This is the reverse of the order in which impairments developed.

The possibility that a masking effect, from sounds generated by tense muscles or changed circulatory conditions, caused the impairments observed in the tests made "during" the Valsalva procedure was controlled as follows: Using himself as the subject, the author obstructed the nasopharynx by inflating a soft rubber balloon in situ after creating positive or negative middle-ear pressure, respectively. The method has been described in a previous paper.[3] The thresholds obtained with respiratory and circulatory strain thus relaxed were practically the same as before inflation of the balloon, showing that the hearing changes were actually caused by the altered middle-ear pressure.

The possibility exists that a slightly different placement of the audiometer receiver was in part responsible for some of the shifts in hearing acuity, especially for the high tones whose wave lengths in air are so short that standing waves may arise in the space between the receiver and the tympanic membrane. Realizing this possibility, care was taken always to replace the receiver in the exact position it

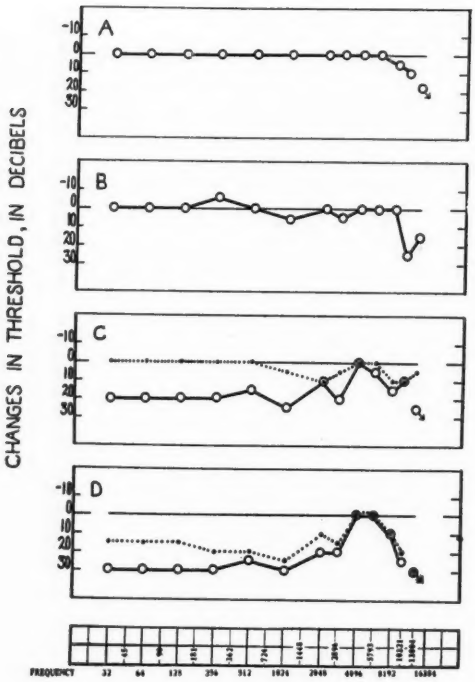

Fig. 2.—Changes in thresholds of hearing with different degrees of negative pressure in the middle ear. The arrows pointing downward in this and in Fig. 3 indicate that the frequency, which was heard originally, could no longer be perceived at the maximum intensity produced by the audiometer.

had been for the earlier tests of the experiment, and in some of the later experiments the receiver was carefully kept in the same position throughout the repeated tests of thresholds of hearing. Certainly slight differences in the position of the receiver cannot explain the amount of the changes for the high tones with negative middle-ear pressure, nor the regularity with which improvement of thresholds for the highest tones occurred with positive pressure and impairment with negative pressure.

Between openings of the eustachian tube the pressure in the middle ear is probably continuously changing, so that complete equalization with atmospheric pressure exists only as a transient stage. The results of hearing tests made after reducing the resistance of the eustachian tube to the passage of air, by shrinkage with ephedrine applied to the nasopharyngeal orifice,[3] indicate that the middle-ear pressure is usually slightly below the atmospheric. Usually the thresholds for very high tones improved slightly in comparison to the preshrinkage level; they never became impaired.

A soft, low-pitched tinnitus was often noted during the experiments with positive pressure, and a faint, high-pitched tinnitus was occasionally remarked upon by some of the subjects during negative pressure. For all observers the tinnitus ceased immediately when pressure equilibrium was established. The amount of the impairment of thresholds shows no relation to the presence or absence of tinnitus or to the subjective impression of its loudness when present. It seems improbable that "masking" by tinnitus can explain any of the observations in these experiments.

Vertigo was reported on three occasions by two test persons in the procedure termed "during positive Valsalva," and on four occasions by three observers "during negative pressure." Objective observations could not be made because the subject always interrupted the pressure immediately. The vertigo was described as rotatory in character and sudden in onset. No nausea was reported. Repetition of the pressure experiment immediately afterwards in no instance reproduced the vertigo.

DISCUSSION

Observations strictly comparable to the present ones have not previously been reported. The most similar studies are those by Bezold[1, 2] and by Siebenmann,[4] both of whom used the Valsalva procedure with normal subjects but used tuning forks to test hearing acuity. Both of these investigators found that either positive or negative middle-ear pressure caused a marked impairment of thresh-

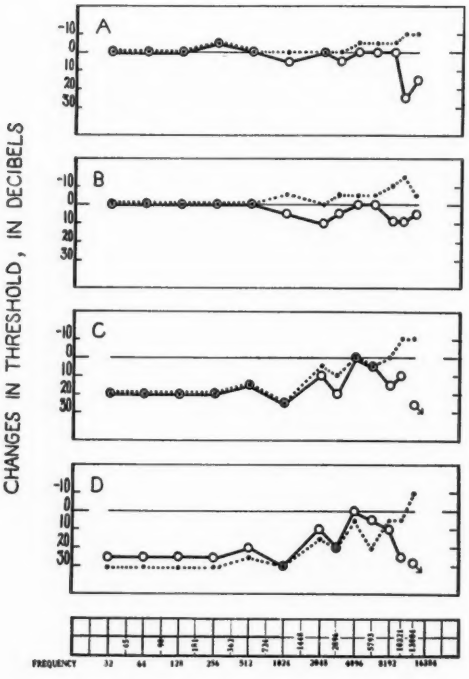

Fig. 3.—Comparison of the changes in thresholds of hearing in one observer, during a two-hour period of observation in which different degrees of positive (dotted line) and negative (solid line) pressure were created in the middle ear by Valsalva's procedure.

olds for the low forks and only a slight impairment for their highest fork (*fis*^{4}, about 2896 cycles per second). Siebenmann extended the range investigated to the highest audible tones by using a Galton whistle, and found impairment for the highest tones with negative pressure but sometimes a slight improvement with positive pressure. It is difficult to express their observations in terms of decibels, but certainly these older studies are in general agreement with the present ones. Bezold's conclusion that hearing for high tones is not significantly affected by pressure changes in the middle ear is not actually in disagreement, since it is easily explained by consideration of the pitch of the tones on which the statement was based.

The animal experiments of Thompson, Howe and Hughson,[5] so far as they can be interpreted in terms of actual hearing, also seem to be in agreement with the present observations. The highest frequency used in their tests was 8192 cycles per second. The animal experiments of Wever, Bray, and Lawrence[6] with positive pressures are also in good agreement.

Secondary changes in the middle ear complicate comparisons of the observations on normal subjects with studies of patients with tubal occlusion, whether chronic or acute, as occurs in aviators whose eustachian tubes fail to open during rapid descents. It seems, however, that different usages of the term "high tones" explain apparently different conclusions in some of the clinical reports.

The observations show that pressure changes in the middle ear can cause "tonal dips." Although the observations afford no explanation for the differential effect on the transmission of neighboring frequencies, they do show that patients with "tonal dips" do not necessarily have a cochlear lesion. It is of interest that hearing acuity was least often affected for the frequency (4096 cycles per second) for which "dips" are most often found in patients.

It has been noted that all the subjects found negative pressure easier to maintain than positive pressure (Fig. 4, *A*, *B*, *C*). The observations confirm the usual idea that the eustachian tube opens more readily during swallowing when the pressure in the middle ear is higher than atmospheric pressure than when it is lower. This observation agrees also with the experiences of aviators, who find it easier to equalize middle-ear pressure during ascent than during descent. It appears that absorption of the contained air is only part of the reason why positive pressure in the middle ear is seldom found in patients.

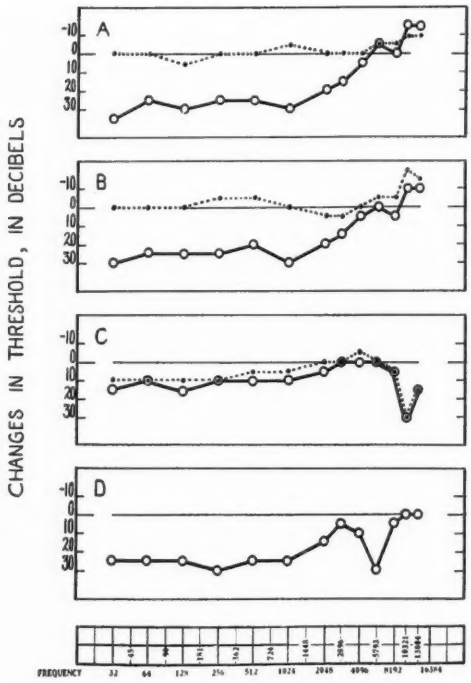

Fig. 4.—*A* and *B*. Examples of changes in thresholds of hearing after sudden release of a marked degree of positive nasopharyngeal pressure. For each, the original audiogram is plotted as the zero line, the changes during the maintenance of nasopharyngeal pressure are shown by the solid line, and the changes after release of the pressure are recorded by the dotted line.

C. An example of the changes in thresholds of hearing after sudden release of negative pressure in the nasopharynx. Comparison of this record with those shown in *A* and *B*, charted by the same method, demonstrates that the eustachian tube behaves differently with respect to positive and negative pressure in the middle ear.

D. An example of a "tonal dip" caused by positive middle-ear pressure. The change for one frequency is much greater than for the neighboring frequencies. A "dip" caused by negative pressure is shown in *C*.

SUMMARY AND CONCLUSIONS

The influence on the hearing of positive and negative pressure in the middle ear, created by Valsalva's procedure, was investigated in normal subjects by means of audiometric examinations.

Both positive and negative pressure, when of marked degree, were found to impair thresholds for the frequencies from 32 to 1024 cycles per second by as much as 20 to 30 decibels, and for the frequencies 2048 and 2896 by about 10 to 15 decibels. Neither type of pressure had much effect on hearing acuity for 4096 and 5793 cycles.

For the higher frequencies produced by the Western Electric Co. 1-A audiometer the effects of positive and of negative middle-ear pressure differed: the thresholds were improved slightly by positive pressure but were impaired by negative pressure. Usually the higher the frequency the greater was the impairment; for 10321 and 13004 cycles it was sometimes 30 to 35 decibels. With negative pressure an increase in the difference from atmospheric pressure increased the impairment for the highest tones, but with positive pressure the improvement caused by a slight pressure was sometimes lost when the pressure was increased.

For both positive and negative pressure the highest tones were the first to be affected as the pressure differential with the atmosphere was increased. With negative pressure the order was then definitely middle tones followed by low tones, but with positive pressures a definite sequence for low and middle tones was not found.

When pressure differences were gradually decreased, the sequence of return of hearing acuity occurred in the reverse order to its loss. Recovery of original thresholds always occurred when pressure equalization was complete, showing that the changes in hearing were caused by the pressure and not by secondary middle-ear changes.

Occasionally, both with positive and with negative pressure, markedly greater impairments were found for one of the high tones than for the adjacent ones. Deliberate attempts to cause such "tonal dips" were not successful.

A faint tinnitus was sometimes noticed with both types of pressure, more often with positive pressure, with which it was described as low-pitched, while with negative pressure it was described as high-pitched.

Vertigo was reported on a few occasions with both types of pressure.

Equalization of middle-ear pressure with the atmospheric pressure was accomplished more easily with positive than with negative pressure. Positive pressure tended to become equalized even without swallowing, but sometimes the negative pressure created by the Valsalva procedure was not changed even by several forceful swallowing acts.

Different usages of the term "high tones" account for the divergent conclusions reported by previous investigators of the effect of middle-ear pressures on hearing.

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XCIII

THE SUBLABYRINTHINE EXENTERATION OF THE PETROUS APEX*

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The accepted surgical approaches to the petrous apex, as described by Eagleton, Frenckner, Kopetsky and Almour, Lempert, Lindsay, and Ramadier, have all reached the antelabyrinthine portion of the petrous pyramid by routes superior to the otic labyrinth. The literature contains only occasional mentions of infralabyrinthine drainage of the petrous apex although infralabyrinthine cellular leads into the petrous apex are a recognized anatomical fact. Surgically these infralabyrinthine leads have usually been drained from the hypotympanum after performing a radical mastoidectomy. Dearmin[1] observed that in some temporal bones it is possible to drain the petrous apex posteriorly from beneath the labyrinth by dissecting along the cerebellar dura and the inner table of the posterior cranial fossa. Repeated anatomical dissections at the University of Michigan have demonstrated that sublabyrinthine leads to the petrous apex may be followed by the continued mesial dissection posterior to the facial nerve from the medial wall of the mastoid tip. In temporal bones where there is no cellular lead to the petrous apex it has been possible to dissect along the same anatomical pathway and thereby create an opening into the petrous apex. Through this sublabyrinthine approach it is possible to exenterate the cellular structure of the petrous apex by a sublabyrinthine route without performing a radical mastoidectomy (Fig. 1).

The surgical procedure of the sublabyrinthine petrous apicectomy begins at the completion of the simple mastoidectomy. The steps in the operation are: First, the delineation of the landmarks in the medial wall of the simple mastoidectomy cavity and the exenteration of the infralabyrinthine cells; second, the dissection of the opening into the center of the petrous apex; and finally the removal of the cellular structure of the petrous apex. This procedure may

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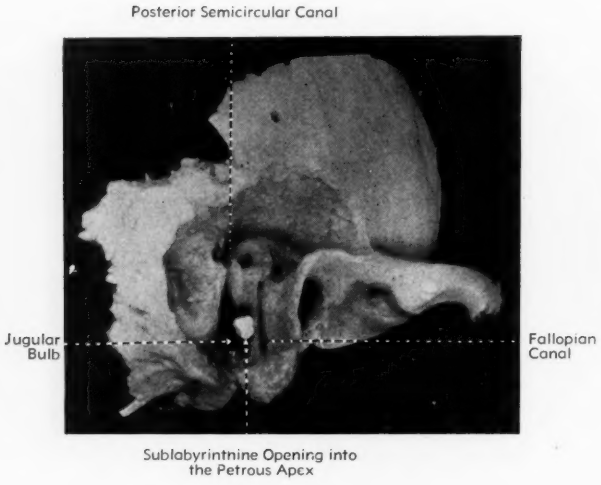

Fig. 1.—The sublabyrinthine opening into the petrous apex is accessible through the medial wall of the simple mastoidectomy cavity, providing an opening for adequate drainage and exenteration of the petrous apex, and not disturbing the contents of the middle ear. The opening passes anteromedially posterior to the facial nerve, under the posterior semicircular canal, parallel to the basal turn of the cochlea, and into the center of the petrous apex.

be performed with safety if each of the anatomical landmarks is positively identified.

ANATOMY

There are four anatomical regions involved: First, the medial wall of the simple mastoidectomy cavity; second, the infralabyrinthine pneumatization; third, the area posteroinferior to the cochlea through which entrance is gained into the petrous apex; and finally, the antelabyrinthine portion of the petrous pyramid.

The anatomical landmarks on the medial wall of the simple mastoidectomy cavity are the semicircular canals, the sigmoid sinus, the digastric crest and the vertical course of the facial nerve (Fig. 2). These structures bound a somewhat quadrilateral portal through which the infralabyrinthine cellular structure may be exenterated. In this procedure the most important landmark is the posterior semi-

circular canal, for, as this canal curves medially and forward to the vestibule, its ampullated end is just medial to the superior end of the vertical course of the facial nerve. The posterior semicircular canal may therefore be followed medially as a reliable guide and injury to the facial nerve thus be avoided.

The infralabyrinthine cellular development may be the result of pneumatization from the floor of the mastoid cavity (retrofacial cells) or from the floor of the middle ear (hypotympanic cells). The development of the retrofacial cell group is directly proportional to the distance between the sigmoid sinus and the facial nerve. In a pneumatic mastoid when the distance between the facial nerve and the sigmoid sinus is minimal, the infralabyrinthine area is more likely to be pneumatized from the hypotympanum.

The infralabyrinthine cells are in relationship with many structures. Anterolaterally from without inward, along the axis of the petrous pyramid, these cells are in relationship with the facial nerve, the hypotympanum, the basilar whirl of the cochlea, and the carotid artery (Fig. 4). Superiorly from without inward are the posterior semicircular canal, the vestibule, the aqueduct of the cochlea, the basilar whirl of the cochlea and internal auditory canal (Fig. 3). Posteromedially and inferiorly are the sigmoid sinus and the cerebellar dura. Medially these cells may extend over the dome of the jugular bulb to come into intimate relationship with the internal auditory canal, they may extend under the jugular bulb toward the occipital condyle, or they may extend anteromedially behind and below the cochlea between the jugular bulb and the carotid artery to pneumatize the antelabyrinthine portion of the petrous pyramid (Fig. 3). Such an infralabyrinthine lead into the petrous apex is more likely to develop from the hypotympanic cells than it is from the retrofacial cells.

The position of the opening into the petroux apex is best visualized by the examination of a transverse section of the petrous portion of the temporal bone through the jugular bulb and the carotid artery. This section reveals a constant area of cellular or cancellous bone postero-inferior to the basal turn of the cochlea (Fig. 3). It is the removal of this bone which creates the opening into the petrous apex. This opening is best described as being in the form of a canal which begins laterally just medial to the ampulla of the posterior semicircular canal (Fig. 1) and extends postero-inferior to the basal turn of the cochlea and inferior to the internal auditory canal into the center of the petroux apex. In this course the canal passes between the jugular bulb and the vertical portion of the carotid artery

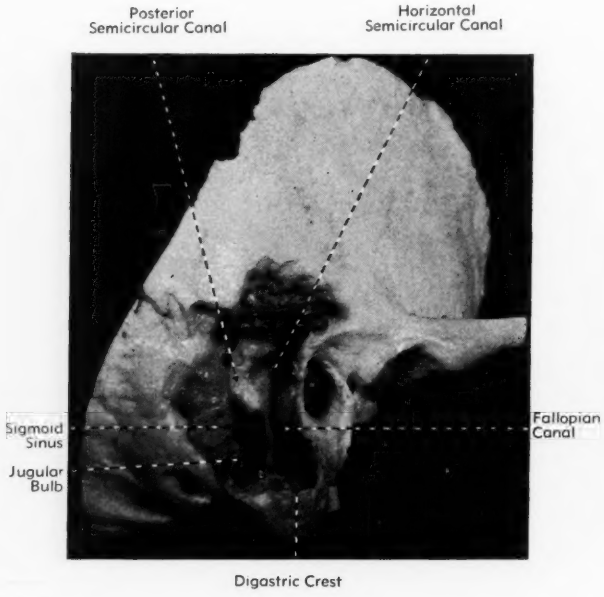

Fig. 2.—The semicircular canals, the fallopian canal, the digastric crest, and the sigmoid sinus are the important landmarks on the medial wall of the simple mastoidectomy cavity. The posterior semicircular canal provides a reliable guide in dissecting posterior and medial to the facial nerve.

and parallel to the posterosuperior surface of the horizontal portion of the carotid artery. Thus in creating the canal the almost invulnerable wall of the basal turn of the cochlea may be safely followed as a reliable guide from the infralabyrinthine portion of the petrous pyramid into the antelabyrinthine portion of the petrous pyramid (Fig. 4). The aqueduct of the cochlea is the most vulnerable structure along the course of the canal. Superiorly the aqueduct of the cochlea is safely enclosed in enchondral bone (Fig. 3). Posteromedially the canal turns downward to empty into the posterior cranial fossa, in the region of the inferior petrosal sinus, 1-3 mm. above and medial to the jugular bulb. It is in the more vertical course that the aqueduct of the cochlea is likely to be injured if the dissection is directed posteriorly along the inner table of the posterior cranial fossa. To avoid this injury and to avoid injury to the jugular

bulb and the carotid artery, all curetting is done along the enchondral bone of the basal turn of the cochlea and the internal auditory canal. The direction of the canal into the petrous apex is horizontal and in the long axis of the temporal bone.

The most variable factor in the infralabyrinthine area is the position of the jugular bulb. In nonpneumatized pyramids the jugular bulb may be superficial and high, in contact with the cochlea and the fallopian canal and even project superiorly above the inferior surface of the internal auditory canal, blocking the infralabyrinthine approach. In a well pneumatized specimen there may be a distance of 5-7 mm. between the jugular bulb and the cochlea, room for a very adequate opening into the petrous apex. If the infralabyrinthine cells are found to be well developed, the jugular bulb must necessarily be placed inferiorly, permitting the sublabyrinthine entrance into the petrous apex. It is my opinion that such a position of the jugular bulb will exist in any temporal bone in which there is pneumatization of the apex.

The antelabyrinthine portion of the petrous pyramid may be composed of either cancellous or cellular bone. The greater mass of the cancellous or cellular structure is posterior to the horizontal portion of the carotid artery, and there is only a small amount of bony structure above and below the artery. (Figs. 4 and 5). The petrous apex may be arbitrarily divided into superior and inferior portions. The superior half of the petrous apex is limited laterally by the cochlea and the internal auditory canal, while the inferior half is limited laterally by the jugular bulb and the vertical portion of the carotid. The sublabyrinthine canal provides entrance directly into the center of the body of the petrous apex. From this point of vantage in the center, it is possible to exenterate the cellular structure in both the superior and inferior halves of the apex.

TECHNIQUE

The delineation of the medial wall of the simple mastoidectomy cavity and the exenteration of the infralabyrinthine cells. The dissection on the medial wall of the simple mastoidectomy cavity is begun by the denuding of the posterior semicircular canal. This denudation should be carried well forward on the inferior surface of the labyrinth to the ampulla of the canal. The dissection should follow the medial and inferior surface of the canal in order to avoid injury to the facial nerve. The digastric crest should then be clearly defined, and the dissection should be carried well forward on its superficial and medial surface. The retrofacial cells may then be

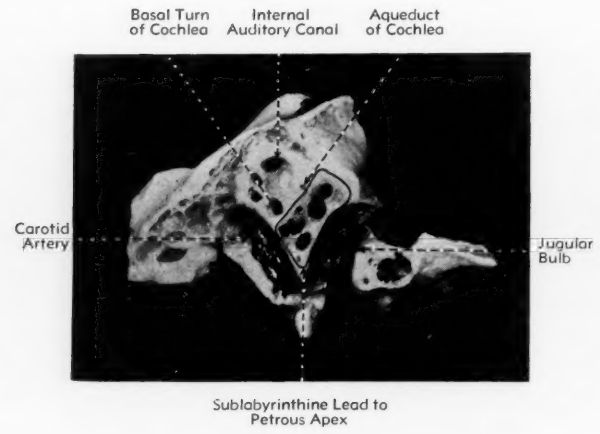

Fig. 3.—A transverse section of the petrous pyramid through the jugular bulb and carotid artery reveals a constant area of cancellous or cellular bone postero-inferior to the basal turn of the cochlea and inferior to the internal auditory canal. This bone may be removed, creating an opening into the petrous apex.

removed, curetting parallel to the sigmoid sinus. As the dissection approaches the facial nerve, it is unsafe to assume that the posterior border of the facial nerve is identified by the point where the digastric crest meets the posterior wall of the external auditory canal, for in a well-pneumatized mastoid this posterior canal wall may be placed anteriorly and as a result the facial nerve may be situated in a shelf of bone protruding into the cavity (Fig. 2). It is safer to regard the course of the fallopian canal as originating above, just superficial to the ampulla of the posterior semicircular canal, and from that point coursing downward and backward in the same plane with the digastric crest. As the posterior semicircular canal and the digastric crest identify the depth of the facial nerve, the exenteration of the infralabyrinthine cells may be accomplished with safety.

The dissection of the infralabyrinthine cells is begun along the sigmoid sinus, curetting medially to clearly identify the jugular bulb. When pneumatization extends over the dome of the jugular bulb, the cells may be dissected to its medial surface without injury to the aqueduct of the cochlea. By dissecting anteriorly the posterior hypo-

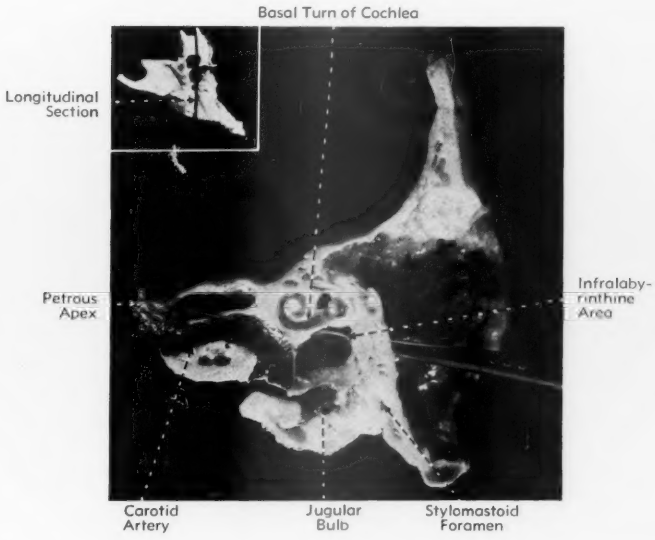

Fig. 4.—A vertical longitudinal section of the petrous pyramid through the basal turn of the cochlea demonstrates that the enchondral bone of the basal turn of the cochlea will serve as an infallible guide in dissecting from the infralabyrinthine area into the antelabyrinthine portion of the petrous pyramid. The tip of the curet may be directed downward to remove the cellular structure posterior and inferior to the carotid artery.

tympanic cells are exenterated through an opening medial to the defined depth of the facial nerve (Fig. 4). Passing a probe into the middle ear through a myringotomy opening will help to identify the mesial wall of the middle ear and the basilar whirl of the cochlea.

Dissection of the opening into the petrous apex. The basal turn of the cochlea is the most important landmark in the depth of the infralabyrinthine area at the beginning of the canal into the petrous apex. The basal turn of the cochlea, beginning at the round window, and the jugular bulb are at about the same depth (Fig. 4) while the vertical course of the internal carotid artery is on the anteromedial aspect of both. By directing the curet anteriorly and medially on a horizontal plane with the inferior border of the posterior semicircular canal, the anterior cutting edge is felt to engage the hard enchondral bone of the basilar turn of the cochlea. As long

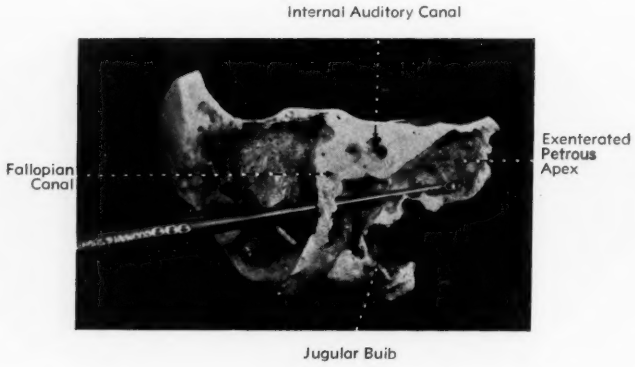

Fig. 5.—Vertical longitudinal section of the petrous pyramid posterior to the carotid artery demonstrates that both the superior and inferior portion of the apex may be exenterated by the sublabyrinthine approach.

as the curetting is horizontal, anterior, and high, the dissection is guided by this almost invulnerable wall of the cochlea and the internal auditory canal. Danger to the jugular bulb, the aqueduct of the cochlea and the carotid artery is thereby avoided. The basal whirl of the cochlea may then be followed as an infallible guide from the infralabyrinthine portion into the antelabyrinthine portion of the petrous pyramid (Fig. 4). Once an opening has been gained into the antelabyrinthine portion of the petrous pyramid, the opening may be enlarged by curetting in all directions. All cancellous bone may be removed from the plates over the jugular bulb and the carotid artery and from the basal turn of the cochlea. To avoid injury to the aqueduct of the cochlea, dissection should not be carried posterior to the dome of the jugular bulb (Fig. 1). When the canal into the petrous apex has been enlarged it should provide an opening 4-7 mm. in all diameters. In curetting anteromedially, with the curet resting against the inferior surface of the internal auditory canal, the curet is felt to slide medial to the hard enchondral bone of that structure and enter the softer bone of the petrous apex.

The exenteration of the cellular structure of the petrous apex. The direct entrance thus created into the center of the petrous apex permits exenteration of the cellular structure. The landmarks which guide the curettage of the petrous apex are the anterior surface of

the internal auditory canal, the jugular bulb, the carotid artery and the inner table of the middle and the posterior cranial fossae. When the curet is in the center of the petrous apex, the shaft is directly posterior and parallel to the horizontal portion of the carotid artery (Fig. 4). All curetting is then directed away from the carotid artery and toward the inner table of the posterior cranial fossa. The cellular structure is first removed from the superior half of the petrous apex extending from the internal auditory canal to the petrous tip. Secondly, the cellular structure is removed from the inferior half extending from the jugular bulb to the tip. Although the exenteration of the apex is largely directed by palpation, the anatomy of the antelabyrinthine portion of the petrous pyramid is sufficiently constant and the sense of touch is sufficiently delicate that the procedure may be completed with safety (Fig. 5).

Although there can be no fixed angulation for the curet, the margin of error is minimized if, in entering the apex, the curet is directed anteromedially in a horizontal plane along the inferior border of the posterior semicircular canal and the basal turn of the cochlea (Fig. 4). Once the apex has been entered, the tip of the apex may be reached by dissecting upward and medially toward the frontal eminence of the opposite side (Fig. 4).

SUMMARY

1. The sublabyrinthine excavation of the petrous apex is presented as a method of performing an intrapetrosal petrous apicectomy without performing a radical mastoidectomy.

2. The anatomical landmarks directing the procedure are described.

3. The technique of the operation is outlined with a view to identifying and preserving all structures which are not to be removed with the cellular structure.

4. The applicability of the approach is evaluated and is found not to be dependent upon a pneumatized infralabyrinthine lead to the petrous apex.

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XCIV

EARACHE: THE COMMON CAUSES AND PROBABLE NERVE PATHWAYS*

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In dealing with earache it is always well to keep in mind that it is deceptive and occasionally treacherous, because the severity of the pain may bear little or no relation to the gravity of the lesion which causes it. All ages are affected, so that it is essential to diagnose the cause and distinguish the urgent from the less serious type, sometimes with little or no history from the patient. For instance, a furuncle or boil in the external canal, although not as a rule dangerous, may cause excruciating pain for a few days, whereas a dull earache which is more of a discomfort than an actual pain may possibly be the only suggestive sign of a mastoid infection bordering on a meningitis. The term earache usually suggests a lesion in or about the ear, and one is apt to forget that the pain may be due to any one of many extrinsic causes, hence the terms "referred pain" and "reflex otalgia."[1]

Irritation of the sensory nerves of the ear in regions far remote from this organ sometimes causes severe pain. This type of earache, without any affection of the ear itself, is called reflex otalgia. In order to understand the mechanism and to interpret the significance of reflex or referred pain, it is necessary to know not only the nerve supply of the ear, but also to what other organs the same or very closely related nerves are distributed.

A number of theories have been proposed to explain the phenomena of referred pain in the body, but none has been proven. In the case of pain referred from an abdominal viscus to the abdominal wall, it is likely that some reflex connection exists between the afferent nerves lying within the (anatomic) sympathetic system and the nerves of the cerebrospinal system. This connection may lie within the spinal cord or in the posterior root. It is probable also that pain can be referred from one branch of a somatic nerve to another of the same nerve, though the exact mechanism of this

*Read before the McGill Reporting Society, March 23, 1942.

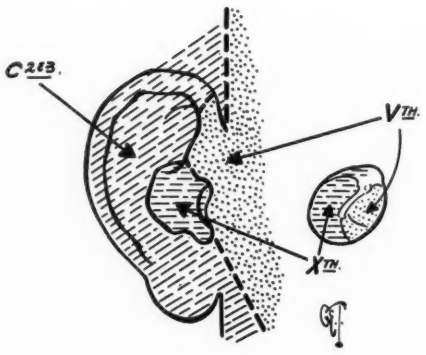

Fig. 1.

reference is unknown (i.e., shoulder pain from irritation of the phrenic nerve fibers in the diaphragm). It is thought that most otalgias arise in this manner, and that it is unnecessary to include the sympathetic and parasympathetic nerves in the reflex pathways. For example, ear pain from the teeth and tongue in all probability travels by way of the fifth cranial nerve, ear pain from the tonsils and eustachian tube via the ninth nerve and ear pain from the throat and larynx through the tenth nerve (Fig. 3).

Some knowledge of the sensory innervation of the ear is necessary in order to understand the various pathways along which these impulses may travel. Briefly, the nerves of sensation to the external ear are as follows: Less than the anterior half of the auricle and the superior and anterior walls of the external auditory canal are supplied by the auriculotemporal branch of the third division of the fifth nerve. Practically the whole surface of the auricle including the helix, antihelix and lobe receives its sensory innervation through the auricular branches of the great auricular and the small occipital nerves from the second and third cervical segments, with an occasional twig from the great occipital nerve.

Although some of the older anatomy texts state that the whole upper half of the auricle as well as the anterior part is innervated by the auriculotemporal branch of the fifth, the examination of patients following surgical section of the trigeminal root indicates that only a small portion of the helix is usually supplied by the auriculotemporal branch of the fifth.

The inferior and posterior walls of the external auditory meatus and concha are supplied with sensation by the auricular branch of the vagus, commonly known as Arnold's nerve. "The ganglionic representation of the sensation of the auricle and the external auditory canal can be divided between the Gasserian ganglion in front, the second and third cervical ganglia behind, and the jugular ganglion of the vagus nerve in between".[2]

There is some anatomical evidence of a sensory branch from the seventh nerve supplying the concha and posterior surface of the auricle as suggested by Hunt.[3] Herpetic eruptions of the concha and external canal are considered to be due to affections of the geniculate ganglion. If this is correct then one must include the geniculate ganglion with the other ganglia concerned with the innervation of the external ear.

The tympanic membrane or drum is one of the most sensitive structures of the body. According to Wilson,[4] the nerve fibers end free between its epithelial cells and are regarded as specific pain sense organs. The anterior portion of the drum is supplied by the auriculotemporal nerve and the posterior portion by Arnold's nerve. The inner surface of the tympanic membrane receives its sensory supply also from the tympanic nerve, a branch of the ninth cranial nerve.

The sensory nerve supply of the middle ear is in the form of a plexus which lies on the promontory or medial wall of the tympanum and is formed by nerves derived from the tympanic branch of the glossopharyngeal (Jacobson's nerve), and receives also a branch from the geniculate ganglion of the facial and the caroticotympanic branches from the carotid sympathetic plexus. The lesser superficial petrosal nerve to the otic ganglion appears to be mainly a direct continuation of the tympanic branch of the ninth (Jacobson's nerve). The skin overlying the mastoid is innervated by the mastoid branches of the great auricular and the small occipital nerves, while the cells in the mastoid process receive their sensory supply through a mastoid branch of the tympanic plexus.

By referring to Fig. 2 the possible course of the common otalgias can be traced and the cause in a great many cases found. Earache is a general term but on questioning it is often possible to ascertain whether the pain is in front of the ear, within the ear itself, or in the region behind the ear. Irrespective of where the pain is situated, one automatically thinks of the distal sensory supply of the fifth, seventh, ninth and tenth nerves or the cervical roots although the

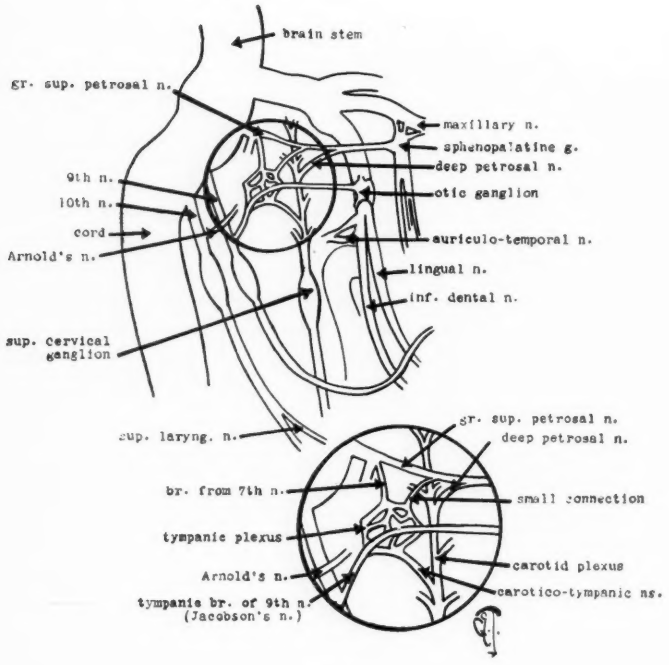

Fig. 2.

peripheral lesion itself may not be painful. Strangely enough the converse does not hold, as lesions in the ear very rarely produce pain in these distal areas. A possible exception might be mentioned although there is no associated pain. Every otologist on swabbing or mopping out the external meatus with a cotton-tipped applicator has often caused the patient to cough. This happens frequently when the posterior canal wall or the corresponding half of the drum is touched. The probable connection is through Arnold's nerve by way of the vagus to the diaphragm stimulating the cough reflex.

As mentioned above, otalgia is caused by an affection of the sensory nerves supplying the organ of hearing. In the same manner earache may occur from an infection in the sinuses, especially in the posterior ethmoidal and sphenoidal. The pathway in this case may possibly be through the sphenopalatine ganglion, along the great superficial petrosal nerve to the geniculate ganglion, which gives off

a sensory branch to the tympanic plexus. It is surprising, however, that pain in the ear is not common in acute sinusitis when one considers the wide distribution of the fifth nerve.

Keeping in mind that the average physician has not the time nor inclination to remember details of a special nature, particularly nerve distributions, the scheme presented in Fig. 3 is shown. It has been used by the writer to impress on students the common causes of reflex otalgia. The simplicity of the diagram is obvious, as all parts affected begin with the letter T.

The most frequent cause of otalgia is of dental origin. More than one-half of all cases are due to dental abnormalities and the patients usually complain that the pain is steady, persistent and worse at night.[5] "The teeth must, therefore, be carefully examined in every case; if they are diseased it may be assumed that they are the probable cause of the otalgia." Those in the lower jaw, particularly, furnish the greater number of cases. Besides caries it must be remembered that an unerupted wisdom tooth is occasionally the cause of an intractable earache. This is easily shown by x-ray examination. "It is probable that the rubbing of their ears by infants cutting teeth is due to some referred sensations in the ears."[6] "If symptoms of a pharyngeal or laryngeal affection are present, we must ascertain by careful examination whether the pain in the ear is not due to these conditions."[7]

Lesions on the side of the tongue, particularly the anterior two-thirds, caries of the lower teeth, inflammatory conditions of the salivary glands, disease of the temporomandibular articulation, may cause pain which is reflected along the auricular branch of the auriculotemporal nerve. This nerve also supplies the parotid gland. It is not uncommon to see an ulceration on the anterior part of the tongue causing pain in front of the ear, while an affection in the posterior third of the tongue causes a severe ache within the ear itself. This is easily explained by referring to Fig. 2, and is due in the first case to involvement of the nerve endings of the auriculotemporal through the lingual branch of the trigeminus nerve, while in the second case the glossopharyngeal is irritated and the pain is referred to the tympanic plexus by way of its tympanic branch. The lingual nerve also innervates the submaxillary and lingual salivary glands, so that a stone in Wharton's duct might easily cause pain in front of the ear.

Patients suffering from acute and chronic affections of the tonsils, tonsillitis, peritonsillar abscess (quinsy) frequently complain

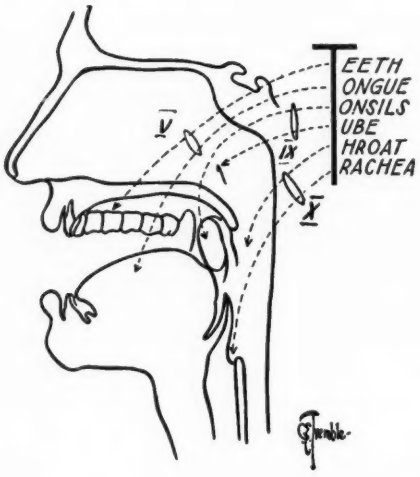

Fig. 3.

of otalgia. "For a few days after tonsillectomy there may be a complaint of earache, and this is felt as a rule only during swallowing."[6] Lesions of the palate or the pharynx, especially in the neighborhood of the orifice of the eustachian tube, often produce pain deep in the ear and occasionally growths of the tongue and tonsil give rise to earache as their first and only symptom. The glossopharyngeal nerve is the one involved in these cases. Ulcerative conditions, such as malignancy or tuberculosis at the entrance of the larynx or upper part of the trachea, may cause reflex otalgia due to irritation of the superior laryngeal branch of the vagus. This nerve "is also used as a guide for nerve block in dysphagia"[8] particularly in tuberculosis of the larynx. On inserting the needle through the thyrohyoid membrane just below the greater cornu of the hyoid bone the internal branch of the superior laryngeal nerve is located when the patient experiences a sharp pain shooting up to the ear.[9] Here again the impulses evidently pass upward through the vagus and Arnold's nerve to the posterior part of the drum and external canal. "It is not often that one finds ear pain in any of the acute inflammatory reactions of the nasal air cells. The extensive nerve association of the fifth nerve with ear, intranasal structures and sinuses makes one wonder why ear pain is not more common than it is."[10]

Infected lymph glands of the neck, goiter, and caries of the cervical vertebrae are rarer conditions causing reflex pain in the ear which do not concern us in this paper. Neurotic women sometimes complain of earache without any apparent reason. The pain associated with migraine is at times localized to the ear.

SUMMARY

Earache may be due to intrinsic or extrinsic causes. By intrinsic we mean organic lesions within the external auditory canal, the middle ear, or mastoid cells, giving rise to pain in the ear. The condition can usually be seen at once by means of the aural speculum or otoscope. Extrinsic causes are those which produce pain in the ear reflexly by lesions remote from the ear itself. The drum in these cases shows little or nothing, usually a normal picture. Ulcerations and disease about the head and neck are responsible for the majority of cases of reflex otalgia. "The source of these pains is almost always explained by examining (1) the buccal cavity, (2) the nasopharynx, and (3) the perilaryngeal region."[11] Of these the commonest are affections of the teeth, tongue, tonsils, tubes, throat and trachea.

The prognosis of otalgia depends on the gravity of the condition causing the earache, and whether or not it is amenable to treatment.*

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XCV

RECENT ADVANCES IN OTOLARYNGOLOGIC ALLERGY*

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Since the publication of "Allergy of the Nose and Paranasal Sinuses" by Hansel in 1936,[1] more than 300 articles have appeared in the literature on various phases of allergy as related to otolaryngology. From this list those which appear to be the most important and significant have been selected for review.

The following subjects exemplify the extent of involvement and the importance of the diagnosis and treatment of allergy in the field of otolaryngology: Allergy of the mouth and oropharynx, the larynx, the esophagus, the cranial nerves, the eustachian tube and the auditory nerve, allergy of the parotid and submaxillary glands, atypical nasal allergy, postnasal rhinorrhea, cytologic studies of the nasal and bronchial secretions, pathology, bacteriology, x-ray, allergy and tonsillectomy, allergy and sinusitis in children and adults, bronchoscopy in asthma, asthma and sinusitis, bronchiectasis and allergy.

In the diagnosis and treatment of allergy of the nose and paranasal sinuses there should be a correlation of the following fundamental or basic factors: (1) the clinical history, (2) the symptoms, (3) the local changes, (4) the cytology of the secretions, (5) the x-ray, (6) the bacteriology, (7) the pathology, and (8) the allergic investigation. And yet, a review of the many papers presented on this subject shows that there is a striking lack of this correlation. One of the most simple and important of these diagnostic procedures is the determination of the cytologic response in the secretions, but in my opinion, this is sadly neglected.

Allergy of the Parotid and Submaxillary Glands. It has been suggested by Rowe[2] that allergic involvement may occur in any of the endocrine or secretory glands. On several occasions we have noted the occurrence or the history of occurrence of unilateral or bilateral swellings of the parotid glands in association with other manifesta-

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tions of allergy, especially angioneurotic edema and urticaria. Rowe has also noted involvement of the submaxillary gland. In our observations, however, we did not recognize the parotid symptoms as the result of allergic involvement until recently, when a patient presented bilateral parotid swelling in association with angioneurotic edema and urticaria. A mucous plug expressed from Stenson's duct revealed a large number of eosinophiles and no neutrophiles. In another case, a patient with nasal allergy, associated with eustachian tube involvement and deafness presented recurring swellings of the right parotid gland. Eosinophiles were noted in the parotid secretion. A third patient had recurring swellings of both parotid glands, mostly the right; he also had nasal allergy and gastrointestinal symptoms. In this case it was noted that sometimes only the upper or lower part of the gland was involved. Eosinophiles were present in the duct secretions.

It is interesting that the demonstration of eosinophiles and also Charcot-Leyden crystals in the parotid secretion were noted by Kussmaul[3] in 1879. In his patient a plug of thick secretion was expressed from the duct and following this there was a gush of clear secretion. In order to demonstrate eosinophiles in the parotid secretion an effort should be made to obtain a mucous plug, for the watery secretion may contain very few cells because of their high dilution. This is also true of watery nasal secretion. A case similar to that of Kussmaul's was reported by Ipscher[4] in which the involvement occurred in the submaxillary gland. Von Reuss[5] noted the occurrence of parotid swellings with the menses in a patient who also showed a blood eosinophilia. Parotid swellings associated with attacks of abdominal colic have also been reported. Londe and Pelz[6] noted the appearance of a unilateral swelling of the parotid gland associated with abdominal colic. These were undoubtedly cases of food allergy.

A most comprehensive report on this subject has been presented recently by Pearson.[7] His observations were based upon the study of seventeen patients, in eleven of which there was no evidence of secondary infection. In the group of eleven patients, seven were children under 12 years of age. There were seven males and four females.

According to Pearson the swelling may affect one or both parotid or submaxillary glands. The swelling usually develops quickly and is often associated with the ingestion of food. Pain and tenderness may be absent. The saliva is usually clear and sometimes there is little or none. Among the eleven patients observed, bilateral swellings occurred in seven.

The condition of the gland ducts was studied by an x-ray examination following the injection of three to six cc. of lipiodol. In five instances there were small dilatations of the terminal ducts or acini. One patient showed a gross dilatation of the main duct and its branches. In one patient the findings were normal. Pearson suggests that obstruction of the ducts may be the result of spasm or an edema of the lining membrane, occurring as a part of an angioneurotic edema of the whole gland. In those instances in which there was no increase in parotid secretion it is reasonable to assume that the swelling was the result of edema of the ducts.

In several cases there was a definite blood eosinophilia. In one patient a very marked eosinophilia was demonstrated in the parotid secretion. Among the 11 patients the association of the following manifestations of allergy was noted: asthma 4, papular urticaria 2, hay fever 3, nasal allergy 1, angioneurotic edema and urticaria 1, and eczema 1. Five patients gave a positive family history and nine both a positive family and personal history of allergy.

In addition to the above eleven patients, Pearson added a group of six patients in which there was evidence of secondary infection. Three of the patients had papular urticaria. One of the patients was sensitive to pork which also caused vomiting. Three patients gave a family history of allergy. In one instance both eosinophiles and neutrophiles were noted in the parotid secretion.

In 1936 Pearson[8] reported two additional cases of bilateral recurrent swellings of the parotid gland. The first case was that of a woman, aged 52 years, who had almost daily attacks. These symptoms began five years previously following an ethmoidectomy. Later she developed asthma and then the swellings of the parotid glands accompanied the attacks of asthma. The family history of allergy was positive. A blood eosinophilia of 15 to 17 per cent was noted. Eosinophiles were present in the sputum. Skin tests showed positive reactions to horse dander, silk, rabbit hair, orris and dust.

The second case was that of a woman, aged 70 years, who had had asthma for 30 years. The recurrent swellings of the parotid glands had occurred for a period of eight years.

The swellings usually develop rapidly, last about an hour, then subside. Relief may be obtained by expressing a jelly-like mucous plug from the duct. After this there is usually a flow of clear saliva and a subsidence of the swelling. The swellings are painless and there is no pain on pressure. The attacks usually come on while eating. The mouth is usually dry and swallowing is sometimes very difficult.

Although it has been suggested by Pearson that obstruction of the parotid duct is caused by edema of the gland, or by edema, swelling or spasm of the gland duct, there is another mechanism which no doubt plays an important part. It has been shown experimentally that the clear salivary secretion from the gland is the result of parasympathetic activity. Vasodilatation is also the result of activity on the part of this nerve. Neither vasodilatation nor vasoconstriction, however, play a part in glandular secretion. Stimulation of the sympathetic fibers results in the production of a thick viscid secretion concomitant with vasoconstriction. It is possible, therefore, that the occurrence of a thick viscid jelly-like plug in the parotid duct may be the result of excessive sympathetic activity. The same mechanism no doubt plays an important part in the production of various types of secretion in the nose, the sinuses and the bronchi.

Nasal Allergy and Salivary Hypersecretion. Among the atypical types of nasal allergy our attention has been called recently to an unusual syndrome of hypersecretion associated with nasal allergy. Three patients of this type have been observed. The chief symptoms consist of a profuse postnasal discharge and little if any anterior drainage. There is very little sneezing, but some obstruction, and at times the nose seems dry. The postnasal discharge is so profuse that the patient is occupied with constant spitting. An ounce of secretion may be expectorated in one to two hours. The secretion is thin and contains a few mucous shreds. On the whole it appears to consist mostly of saliva. If the secretion came principally from the nose, there should be a profuse anterior discharge. Microscopically the secretion shows a moderate number of pus cells, epithelial cells and small clumps of eosinophiles which are not always present but when found there are enough present to establish the diagnosis of allergy. This syndrome appears to be very closely related to that of allergy of the parotid gland except that in the nasal cases there is hyperactivity on the part of the parasympathetic nerves rather than the sympathetic nerves. In any event there is an imbalance in function in both instances.

Two of the above three patients had had nasal and sinus operations with no relief of the symptoms. One patient had a submucous resection, removal of nasal polyps and bilateral antrum window resections. The nose appeared to be in good condition except for an allergic mucosa. In the other case bilateral antrum windows were resected. They were patent, as no secretion collected in either antrum. The nose was in good general condition except for some bogginess of allergic origin. The third patient had a stuffy, boggy

nose with dryness and a moderate degree of obstruction. No surgical interference had been instituted in this case.

Allergy of the Mouth, the Oropharynx, the Esophagus and the Larynx. It has been reported that the following lesions in the mouth may be manifestations of allergy: cheilitis, aphthous stomatitis, gingivitis, glossitis, urticaria, angioneurotic edema and purpura.

Loveman[9] reported an interesting case of contact stomatitis caused by a cream, containing anethol, used for cleaning an artificial denture. Positive patch skin reactions were noted in the cream.

Hecht and Rappaport[10] noted the occurrence of cheilitis with fixed drug eruption and gastrointestinal allergy from the eosin dye of lipstick.

Watson-Williams[11] reported two cases of purpura with ulcerations of the oropharynx from novarsenobenzol. One patient also had involvement of the esophagus. The skin was likewise involved. The second patient showed lesions of both the mouth and the pharynx. A third patient had purpuric lesions of the nose, the mouth and the pharynx following the ingestion of "Aspro" tablets.

Withers[12] reported a patient with marked dysphagia due to food allergy. It was not determined whether the symptoms were caused by edema or by spasm.

It is generally known that angioneurotic edema of the larynx of allergic origin is not uncommon. In a recent discussion of this disease Miller[13] reports his observations on the mechanism of the spread of edema in this locality by experiments on the cadaver.

Frank[14] noted the occurrence of angioneurotic edema of the larynx caused by a sensitivity to chicle. He calls attention to the fact that laryngeal involvement of this type usually occurs in individuals who have or have had other manifestations of the disease. Jackson[15] has reported that 33 per cent of the patients who have died of angioneurotic edema of the larynx could have been saved by prompt tracheotomy.

Taquino[16] reported the occurrence of laryngeal edema and later stenosis with hyperplasia following the administration of tetanus antitoxin.

Imperatori[17] reported a similar case with bilateral abductor paralysis. There was also edema and later perichondritis of the larynx.

More recently Neffson[18] reported a case of unilateral recurrent paralysis from the same cause. He calls attention to the fact that paralysis of other cranial nerves may occur and gives references to reports of involvement of the optic,[19] the oculomotor,[20] the facial, the auditory[25] and the recurrent laryngeal nerves.[21-24]

Bedell[26] recently noted the occurrence of retinal involvement following the administration of tetanus antitoxin. Plumer[27] reported the occurrence of retinal lesions from food allergy. Prewitt[28] observed a patient with retinal detachment associated with urticaria.

Allergy and the Auditory Nerve. Recent observations have also shown that the cochlear and the labyrinthine portions of the auditory nerve may be affected in allergy. In 1936 Cutter[25] reported a case in which a 14-year-old boy, following the administration of tetanus antitoxin, developed increasing deafness, noises in the ears and double vision. A few days later an audiogram showed a loss of hearing for speech of 40 per cent in the right and 50 per cent in the left ear. Three days later the loss was 61 and 69 per cent respectively. Six weeks later he was subjectively well with no tinnitus or deafness but the audiogram still showed a loss of hearing of 9 per cent in the right and 21 per cent in the left ear.

Although foreign serum appears to be the most common cause of cranial nerve lesions of allergic origin, there is every reason to believe that foods and drugs may cause similar involvements, for they not infrequently cause edema and urticaria of the larynx.

Recently it has been shown that allergy plays a more definite part in the cause of some cases of Ménière's disease, than was formerly believed. Granstrom and Nylen[29] reported the case of a woman, aged 56 years, who had repeated attacks of Ménière's disease associated with palpebral edema and intra-ocular edema. The cochlear and the vestibular functions were normal between attacks. During attacks the symptoms consisted of vertigo, rotary horizontal nystagmus on change of the position of the head and a marked reduction of hearing in the left ear. Jones[30] has reported a case of chronic allergic labyrinthitis caused by the ingestion of milk.

Additional reports on Ménière's disease has been presented by Grove,[31] Skoog,[32] Atkinson,[33] Criep,[34] Furstenberg[35] and Proetz.[36] Grove emphasizes the important point that the disease is frequently accompanied by other manifestations of allergy. He refers to the observations of Vogel[37] who noted that among 83 patients with Ménière's disease, 5 had hay fever, 9 urticaria, 15 nasal allergy and 5 asthma, an incidence of allergy of about 40 per cent. It was fur-

ther stated that no pathologic changes have been demonstrated in the auditory nerve. Among 228 cases of Ménière's disease Dederding[38] recorded headache as the most frequent extra-aural symptom, being present in 91 per cent. It has been pointed out by a number of observers that water retention is associated with the attacks, suggesting thereby the presence of generalized angioneurotic edema.

In a very recent paper Horton[39] reported a series of 49 cases of Ménière's syndrome in which prompt relief was obtained by the intravenous administration of histamine. The dosage employed was 1 mg. of histamine base in 250 cc. of physiologic saline, administered at the rate of 50 to 60 drops per minute. To prevent future attacks a maintenance dosage of 0.1 to 0.2 mg. given subcutaneously two to four times a week was suggested.

Allergy and Sinusitis in Children. During the past few years a number of reports presented on the subject of sinusitis in children have not given due consideration to allergy as an etiologic factor. Crooks,[40] for example, presented his observations on 100 cases advocating certain local and surgical procedures and did not even mention allergy. Likewise Birdsall[41] reported 80 cases which were very adequately studied from every standpoint except allergy. And yet an analysis of his studies reveals that a very large percentage of the patients had allergic involvement of the sinuses.

In the diagnoses of chronic nasal and sinus conditions in children, as in adults, it is of primary importance to differentiate allergy from infection. In an analysis of 455 patients referred to the Pediatric Allergy Clinic of Washington University we found that 341, or 75 per cent, had a definite respiratory allergy. Among the remaining 114 cases, or 25 per cent, there were 44 who had other manifestations of allergy but none in the respiratory tract. Six of these patients had acute or subacute sinusitis which was treated by local conservative methods. The remaining 70 patients had colds, bronchitis and sinusitis. Of this group 20 of 22 had cloudy sinuses as shown in the x-ray films. The antrum window operation was performed on six patients. Among the 341 allergic patients this operation was performed on only nine.

In this entire group it is evident that these are the patients who present the problem of determining chiefly the cause of such complaints as frequent colds, chronic nasal discharge and bronchitis, and in whom it is a question of the differentiation between allergy and infection.

Allergy and Tonsillectomy. Among these patients as well as those with a history or clinical evidence of sore throats, tonsillitis, enlarged cervical glands, otitis media and other infections, the question arises as to the advisability of removal of the tonsils and adenoids. In 1938 we[42] made a special study of this group of candidates for this operation with the idea of determining the incidence of respiratory allergy in 200 cases. Twenty-six, or 13 per cent, were positive. Six patients had hay fever.

In a group of 341 cases of respiratory allergy Hansel[43] found that 54.5 per cent of the patients still had their tonsils and adenoids. Before a diagnosis of respiratory allergy had been made, 45.5 per cent had had them removed. In 7.6 per cent the operation was performed after being under allergic management. Although it is granted that in many of these cases the operation is indicated and is of definite benefit, the important point must be emphasized that allergy should be recognized as a possible cause or contributing cause of the symptoms.

Allergy and Chronic Sinusitis—End Results of Surgical Interference. In a symposium on the end results of sinus surgery presented at the Triological Society meeting in Chicago in 1939, the relative merits of various types of surgical procedures were discussed by Hempstead,[44] Faulkner,[45] Salinger[46] and Arbuckle.[47] Although allergy was considered as an important factor, it received hardly more than passing mention.

Hempstead presented his observations on the study of 385 patients treated by the conservative type of intranasal operation on the antrum. Only two patients were subjected to the Caldwell-Luc operation. On the basis of a review of the literature it was shown that among 1,634 patients operated upon by the intranasal method, a good result was obtained in 97 per cent. Hempstead emphasized that when infection complicates an allergic process, a dry antrum cannot be obtained until the allergic conditions has been controlled.

In evaluating the end results of the intranasal operation on the ethmoid, the frontal and the sphenoid sinuses, Faulkner states that a satisfactory outcome is usually dependent upon the performance of a complete operation.

As to the end results of the external operation on the maxillary sinus, Salinger stated that this procedure should be followed by satisfactory results when the preoperative diagnosis is based on sound facts and careful study and when possible allergic factors have been thoroughly investigated and controlled.

In his discussion of the end results of external operations on the frontal, the ethmoid and the sphenoid sinuses, Arbuckle states, "Rhinologists have seen the rise of popularity of surgical treatment of sinal disease on the basis that infection was the underlying cause in all cases. During all this time, in a certain number of cases, relief was not obtained by such treatment. Then they found that many such conditions are not of infectious origin but are allergic. Accordingly, the popularity of operative treatment has decreased almost to the exclusion of certain types of surgical intervention in favor of antiallergic treatment and, occasionally, endocrine treatment."

Arbuckle further calls attention to the rapidly increasing general belief that allergy is a most important factor in the cause of the complaint in well over half of all persons suffering with symptoms of sinal disturbance and that when the condition is correctly diagnosed and properly treated, operation is contraindicated except in instances of infection of a type for the relief of which surgical intervention is indicated; therefore, it is emphasized that the necessity of differential diagnosis between infectious sinusitis and allergic sinusitis is obvious. Arbuckle also emphasized the importance of cytologic examination of secretions in all cases and the necessity of instituting adequate allergic study and care.

In Arbuckle's experience he has noted that, as a rule, when allergic sinuses are operated upon, symptoms disappear for from six weeks to six months, after which the allergic symptoms recur when the patient is exposed to allergens to which he is sensitive. This is in agreement with other observers. He also remarks that he has observed a number of patients with nasal polyps in the early stage of development in whom the polyps disappeared with allergic management and remained absent until this growth was restimulated by an acute infection or an allergic exacerbation. On the other hand, attention is called to the fact that polypoid tissue which has become fibrotic does not disappear with such treatment.

In view of the high incidence of allergy in chronic sinusitis it is evident that the most satisfactory results are dependent upon adequate allergic management. A recent report on allergic rhinitis in which there was cooperation between the rhinologist and the allergist was presented by Woodward and Swineford.[48] This paper illustrates the value of such cooperation. A group of 128 patients was studied. In 61 there were no significant complications. The remaining 67 showed the following: obstruction from the septum or turbinates and chronic ethmoiditis 25; obstruction from nasal polyps 16; pansinusitis and marked mucosal degeneration 9; chronic maxillary sinus-

itis 9; acute and subacute maxillary sinusitis 4; and septal or turbinal obstruction without secondary infection 4.

The opinion was expressed that in general a period of allergic management plus conservative rhinologic treatment should precede surgical intervention. In those few patients with long standing severe asthma, bronchiectasis and physical and roentgenological evidence of chronic sinus infection, they further state that little is to be gained by the postponement of surgical intervention. It was also emphasized that when a major surgical operation is necessary, it should be complete enough to effect a cure of the complications which necessitated it. In the group of 128 patients major surgical procedures were carried out as follows: obstruction and secondary ethmoiditis 25; external operations 18.

An evaluation of the results obtained in 89 cases which were followed up were as follows: 67 were still having nasal symptoms but 58 were from 75 to 100 per cent improved; 18 were 50 per cent improved; while one reported less than 50 per cent improvement.

Bronchoscopy in Asthma. The value of bronchoscopy in the diagnosis and treatment of asthma has been repeatedly emphasized by many observers. The recent contributions of Clerf,[49] Stokes,[50] Andrews,[51] Bozer,[52] Prickman and Moersch,[53] Vadala [54] and others present further observations of importance.

Since dyspnea and wheezing may be the symptoms of allergic as well as nonallergic conditions, bronchoscopy is indispensable in diagnosis in certain types of cases. In the absence of a diagnostic eosinophilia of the bronchial secretions in suspected asthma, bronchoscopy excluding contraindications should always be performed. Even in asthma this procedure is of value in treatment for the aspiration of secretions gives definite relief in certain cases. The aspirated secretions should be cultured, and cytologic studies made. Vaccines can be prepared from them for use in treatment.

Among 140 asthmatic patients selected for diagnostic bronchoscopy by Prickman and Moersch there were 60 who had definite stenosis of one or more bronchi. They state the bronchostenosis is primarily inflammatory and not referable to allergic edema or bronchial spasm. One of the most prominent symptoms of this condition is a severe, persistent and sometimes paroxysmal cough. At first it is impossible for the patient to raise sputum, and when it appears it is profuse, usually mucopurulent and sometimes streaked with blood. Febrile episodes, either with or without preceding chills occurred in 68 per cent of the cases reported. Of the sixty patients 53 per cent

gave a history of pneumonia; 35 per cent of pleurisy. Acute infection of the respiratory tract, therefore, appears to be the important etiologic factor in producing bronchostenosis. Treatment consists in dilatation of the stenosis and aspiration of secretion.

Valada[54] also emphasizes the importance of bronchoscopic examination, aspiration and culture of bronchial secretions in those cases in which the differential diagnosis lies between chronic bronchitis, pulmonary tuberculosis, asthma and bronchiectasis. His observations were directed chiefly to mycotic infections. A number of interesting case reports were presented.

The Sinuses and Asthma. There is no subject in Otolaryngology about which there has been more diversity of opinion than sinusitis and asthma. Harbert[55] has recently expressed the opinion that the differences reported are due to an absence of a control series of cases, accuracy in diagnosis, standardization of cures, observation over an insufficient period of time and finally, failure to consider the allergic process as the primary factor. He calls attention to the high incidence of positive roentgenographic evidence of sinus disease in these cases. He properly emphasizes that these changes are not an indication for operation but merely represent varying degrees of sinus edema. He favors the conservative methods of treatment, paying particular attention to the allergic state and later considering whatever surgical procedures are necessary.

A recent report by Kelley[56] on the sinus changes in asthma is fairly representative. In 100 cases of bronchial asthma, 89 per cent showed x-ray evidence of chronic hyperplastic changes involving one or more of the sinuses. Sixty of the 89 patients showed definite changes in the nose. Twenty-three, or 38 per cent, had nasal polyps.

The following few reports on the results of surgical procedures on the sinuses in asthma present some idea of the marked diversity of opinion as to the final outcome. For example, Jacquelin and Chait[57] selected from 430 asthmatics 48 patients with marked nasal changes and 26 with infectious manifestations and subjected them to various procedures such as submucous resections, removals of polyps, antrum irrigations and sinus operations. A small number of patients improved, some were no better and others were worse.

In considering asthma from the otolaryngologic standpoint Fox and Harned[58] carried out various surgical and nonsurgical procedures on 300 patients. They were divided into a surgical group of 150 and a nonsurgical group of 150. The surgical procedures were only of the radical type. Forty patients had complete exenteration of all

sinuses, 40 had intranasal radical ethmoidectomy and the Caldwell-Luc operation on both sides, 50 had only intranasal radical ethmoidectomy, and 20 had bilateral Caldwell-Luc operations. The results reported were as follows: among those who had total exenteration 60 per cent were cured. In the second group 45 per cent were clinically cured, in the third group 32 per cent and in the last group 60 per cent were cured. In the group of 150 patients treated by various nonsurgical and nonallergic methods the results were not so striking.

In the management of sinusitis in asthma no one has had more experience than Weille[59] who has been working in collaboration with Rackemann on this problem. In a recent report Weille presented his observations on 500 asthmatic patients. Evidence of sinusitis was found in 362, and 223 had nasal symptoms. Sinusitis was slightly more common in intrinsic than in extrinsic asthma. Polyps were present in 211, deviated septum in 92, chronic tonsillitis in 35 and abscessed teeth in 84. Sinus operations were performed on 100 patients as follows: 17 intranasal antrums; 41 radical antrums; 30 intranasal ethmoids; 5 external ethmoids; and 17 sphenoids and external frontals. More or less improvement in the asthma was noted in 66 per cent of those having sinus operations. Improvement was the same in intrinsic as in extrinsic asthma. The higher percentage of improvement in asthma followed conservative nasal surgery rather than very drastic operations. Among 186 patients with chronic tonsillitis 35 had them removed and 25 had improvement in the asthma. Of 84 patients with abscessed teeth 59 had them removed and 39 patients had improvement in the asthma. Among 36 patients subjected to bronchoscopy as a part of the treatment 12 were improved and three were cured. There were 290 patients who had no surgical treatment whatsoever, although many had sinusitis, chronic tonsillitis and abscessed teeth. The proportion of good results in this group treated only medically was about the same as in the group treated surgically.

The observations of Weille are in agreement with those who have had an extensive experience in the management of these problems. It is apparent that the conclusions must be accepted that the sinus disease accompanying asthma is not the cause of the latter but represents a part of the picture of allergic involvement. Although it is generally agreed that certain surgical procedures are indicated in selected cases regardless of whether asthma is, or is not, present, the management of the allergic problem is of primary importance.

Allergy, Sinusitis and Bronchiectasis. Bronchiectasis is not an infrequent complication of bronchial asthma; on the other hand,

chronic bronchitis and bronchiectasis may occur in the absence of definite asthma. Polypoid changes in the nose and paranasal sinuses similar to those noted in allergy cases commonly coexist in cases of bronchiectasis without asthma. Few observers have paid any particular attention to the incidence of allergy in these cases. The changes in the nose and the sinuses and the bronchi have been treated on an infectious basis. Those patients with pathologic changes in the sinuses have been subjected to surgical procedures because of the possible etiologic relationship of the two conditions.

In studying the relationship of sinusitis to bronchiectasis, Walsh and Meyer,[60] noted that among 217 patients, 145, or 66.8 per cent, showed associated sinusitis. Various symptoms and illnesses such as influenza and pneumonia preceded the apparent onset of the bronchiectasis. They observed that the extent of sinus involvement was variable and that there was no relation between the degree of sinusitis and the degree of bronchiectasis. Chronic pansinusitis or gross bilateral infections of the antrum was noted in 78 cases.

Goodale[61] reported similar observations on a group of 75 patients. On the basis of x-ray evidence 46 patients had chronic sinus infection and 29 had negative sinuses. Eighteen of the 29 negative cases, however, gave a history of clinical evidence of sinusitis, either recurrent acute or mild chronic sinusitis. In 11 cases, or 14.5 per cent, sinusitis did not appear to be a factor. Goodale found that the prevalence of sinus infection paralleled the extent of the bronchiectasis, differing in this respect from Walsh and Meyer.

In a more recent paper Goodale[62] reported his observations on 150 cases, stating that about 90 per cent of the cases of bronchiectasis show evidence of sinusitis.

The above-mentioned reports are representative of many observations made during the past several years on the subject of sinusitis and bronchiectasis. The study of the cytology of the secretions for eosinophiles has not been utilized as a diagnostic procedure in these cases nor have they been consistently investigated from the allergic standpoint. There is much need, therefore, for further studies on the possible incidence of allergy in bronchiectasis, as shown by the following reports in which it was considered.

Watson and Kibler[63, 64] noted that bronchiectasis is seldom found without at least some evidence of sinusitis. They also observed that many cases of bronchiectasis had some manifestations of allergy, such as hay fever, asthma, urticaria, eczema or rhinitis. Upon studying the cytology of the nasal secretions, they were impressed with the

frequency of the high percentage of eosinophiles. A similar study of the sputum likewise showed an abundance of eosinophiles in a large percentage of the cases of bronchiectasis. On this basis they began the investigation and treatment of these patients from the allergic standpoint and were able to obtain most satisfactory results.

Thus, a new conception of the etiology of bronchiectasis which makes its prevention and recovery possible has been presented. They found that bronchiectasis may frequently be successfully combatted in its earlier stages if considered a sequela of allergic bronchitis. In view of the fact that many patients with chronic sinus involvement and bronchiectasis seek the warm dry climate of Arizona for relief, the authors have had an unusual opportunity to observe many cases of this type.

They have noted that patients with bronchiectasis frequently have hay fever, eczema, moderate but definite asthma at times, or other manifestations of clinical allergy. Among all patients with bronchiectasis it was observed that in general there was an abnormally high percentage of eosinophiles in the bronchial secretions; that is, 10 per cent or more. In fully 90 per cent of the cases, a definite diagnosis of allergy could be made on the evidence found.

In a recent unpublished presentation on the relation of allergy to bronchiectasis Riecker[65] of the University of Michigan stated that among 127 patients with bronchiectasis, seven had asthma, 63 nasal allergy, 19 both nasal allergy and asthma, and 38 showed no allergy. The incidence of allergy in the entire group, therefore, was about 70 per cent. Among all patients with asthma he found an incidence of bronchiectasis of about 3 per cent.

With the idea of determining the incidence of bronchiectasis in asthma Viswanathan[66] studied 50 consecutive cases of asthma varying from three to ten years' duration. Fourteen patients, or 28 per cent, showed definite evidence of bronchiectasis, six showed saccular dilatation, five cylindric dilatation and three early bronchiectasis with tortuosity of the bronchioles and slight cylindric dilatation.

More recently Woodward and Swineford[48] reported an incidence of bronchiectasis of 12.5 per cent among 128 cases of nasal allergy. Seventy-four of the 128 patients had asthma.

X-ray of Sinuses. Based upon a study of 854 cases, Johnson[67] reports his observations on the value of roentgen examination of the paranasal sinuses. In this group the rhinologist and the roentgenologist agreed as to the presence or absence of sinus disease in 80 per

cent of the patients. A review of the cases in which there was disagreement reveals that paranasal sinus disease was thought to be present in 99 cases on the basis of the roentgenologic findings and in 76 cases because of physical signs. In more than half the roentgenologically positive group, however, there was only slight residual mucosal thickening and these were cases of clinically inactive or cured sinusitis. On the other hand, those cases of sinus disease in which the diagnosis was based on physical findings alone represented, for the most part, examples of mild allergic rhinitis.

In 15.5 per cent or in 132 cases the roentgen findings were compared with the operative findings. In only a few instances was there any disagreement. In this group of patients operative interference was instituted as follows: unilateral maxillary 62, bilateral maxillary 26, miscellaneous individual or combinations of ethmoid, sphenoid and frontal or maxillary 44.

In 163 sinuses showing simple clouding (soft tissues) the operative findings were as follows: normal 4, pus 63, pus and thickened mucosa 67, thickened mucosa 6, polypoid mucosa 15, neoplasm, benign, 1, and neoplasm, malignant, 7. Bone involvement occurred as follows: osteitis 6, osteomyelitis 3, and malignant invasion 5. Among 13 operative sinuses showing clouding with bone destruction the findings were: normal 1, pus 1, pus and thickened mucosa 3, polypoid mucosa 1, malignant neoplasm 7. Among those cases with bone involvement 6 had osteomyelitis and 6 had malignant invasion.

In 37 sinuses showing thickened mucous membranes the operative findings were: normal 3, pus 13, pus and thickened mucosa 15, thickened mucosa 3, polypoid mucosa 2, malignant neoplasm 1. Among three cases with bone involvement one had osteitis and two osteomyelitis.

Johnson concludes that this review of actual experiences with the radiographic approach to the diagnosis of paranasal sinus disease offers convincing proof that such methods are reliable if the limitations of the procedure are clearly appreciated.

According to Macmillan,[68] in chronic sinusitis there is a reaction in the bone in the form of an osteitis. In chronic ethmoiditis there is a sclerosis of the cell partitions. In chronic polypoid ethmoiditis the cell partitions are decalcified.

In the allergic cases the general appearance is one of a lack of contrast. There is no clear-cut detail of blackness to the entire sinus area. The ethmoid cell partitions may be entirely decalcified. The

film may have a milky washed, non-contrasting appearance. There is no reaction in the bone as seen in chronic purulent sinusitis.

Macmillan found that about 20 per cent of the patients complaining of sinus trouble show mucous retention cysts in the antrum. They were unilateral or bilateral.

Pathology of Sinusitis. In a very comprehensive presentation, Semenov[69] discussed the surgical pathology of nasal sinusitis, based on a group of 500 surgical cases. Microscopic examination of the mucosa showed that when thickening in excess of 2 mm. is present, it is associated with deep-seated degenerative changes in 50 per cent of the cases. Of the 500 cases, Semenov classified 72 per cent as purulent sinusitis, and 28 per cent as nonpurulent hyperplastic polypoid and cystic degenerative. Manifest allergic sinusitis occurred in 17 per cent; 35.4 per cent were classified as equivocal allergic cases. These latter cases were considered as latent allergic conditions in which the history was uncertain, vague or incomplete, but in which microscopic examination showed tissue eosinophilia, associated with other histologic findings sufficient to warrant a presumptive diagnosis of latent allergy. Nonallergic inflammation as determined by careful history and examination and histologic preparations was present in 47.5 per cent of the cases. Degenerative changes were more pronounced in allergic sinusitis. He states that hyperplastic sinusitis, especially the bilateral type, is allergic in 70 per cent of the nonpurulent cases.

Tissue cultures revealed a preponderance of streptococci and staphylococci in chronic sinusitis, mixed infection being present in 80 per cent. It was further found that exudative sinusitis usually responded to conservative treatment. Degenerative changes which are irreversible in character required radical treatment. A study of postoperative healing in the paranasal cavities showed that it is accomplished by the formation of a dense layer of white, fibrous connective tissue which epithelizes by an ingrowth of nasal mucosa.

Bacteriology of Sinusitis. Among allergists and bacteriologists a difference of opinion exists as to whether the hyperplastic form of sinusitis is caused by sensitization to air-borne substances and to foods that can be determined by positive skin tests or whether it is caused primarily and solely by infection. Grove and Cook have previously expressed the opinion that hyperplastic sinusitis is caused solely by infection, which produces a special pathologic response conditioned by the allergic constitution of the patient. More recently a further report was presented by Grove and Farrior[70] based on the

study of the bacteriology of 200 operative cases. Preoperative bacteriologic examinations were made of the washings or surface swabs. Both bacteriologic and pathologic studies were conducted on the sinus membranes removed at operation. In Kistner's study of the histology and bacteriology of 400 cases of sinusitis of the hyperplastic or nonsuppurative type he noted that the streptococcus was the predominating organism. A mixture of bacteria, however, was usually found in each case; therefore, pure cultures were seldom noted.

In the group of cases studied by Grove and Farrior, 80 per cent of the patients had asthma. One-half the patients showed one or more positive skin reactions but they were not always clinically significant.

Three hundred and sixty-five cultures were made of washings from the sinuses, over 90 per cent of which came from the antrum. One hundred and thirty cultures were made from the membranes removed from the ethmoid and sphenoid sinuses and 108 from the antrums. Some form of staphylococcus was by far the most frequent organism encountered in the sinus washings as well as in the membranes. Hemolytic streptococci were found more frequently in the antral membranes than in the washings or in the ethmoidal or sphenoidal membranes. Ninety-five per cent of the antral membranes and 80 per cent of the ethmoidal membranes showed bacteriologic evidence of infection or an average of 87 per cent for the total group.

On the bases of these studies Grove and Farrior concluded that chronic hyperplastic sinusitis is caused by bacterial infection and represents a special type of bacterial allergy.

DISCUSSION

In summarizing this review of the recent literature on allergy in Otolaryngology, it is evident that many distinct advances have been made. It has become more and more apparent that in addition to the nose and the paranasal sinuses other important structures in this field are not infrequently involved, such as the salivary glands, the eustachian tube, the internal and external ear, the cranial nerves, the mouth, the pharynx, the larynx and the esophagus. Further studies have been made on bronchoscopy in asthma, the relation of sinus involvement to asthma and bronchiectasis.

On the other hand, in the management of the nose and paranasal sinuses there is a striking lack of the correlation of all the factors which enter into the diagnostic problem. In establishing the diag-

nosis and in making a decision as to possible surgical interference, the following should be considered: the clinical symptoms, the rhinoscopic examination, the cytology of the secretions, the x-ray films of the sinuses, the bacteriology and pathology. If infection is found to be present at the primary investigation it must be determined whether it is acute, subacute or chronic. Not infrequently two to four weeks' observation, especially with repeated cytologic studies, is necessary before a decision can be made.

In the observation of children with frequent recurring colds and bronchitis the otolaryngologist and the pediatrician must determine whether allergy or infection is the cause of the symptoms. Among the allergic patients in this group too many tonsillectomies are unnecessarily performed.

Persistent bronchitis in children, not infrequently in the absence of nasal symptoms, is of allergic origin and often precedes the development of asthma. Chronic disease of the paranasal sinuses in children is often allergic and not primarily caused by infection.

In adults nasal stuffiness and postnasal discharge even in the absence of sneezing may be allergic in origin.[71] Although a submucous resection is sometimes necessary in these cases it may fail to give adequate relief. Allergic management should always be tried before resorting to the use of the cautery or escharotics.

In the management of the typical case of nasal allergy with no complicating infection or polyposis, surgical interference, other than a possible submucous resection, is not indicated. An x-ray examination of the sinuses in those cases, however, usually shows some degree of cloudiness which from the roentgenologist's standpoint is generally reported as sinusitis. These changes are often transitory in nature and they are the rule rather than the exception.

In those cases showing varying degrees of polyposis in the nose as well as edematous and polypoid changes in the sinuses the question arises as to the degree of secondary infection or suppuration and as to what type of surgical interference should be undertaken. If infection is present it must be determined whether it is acute, subacute or chronic. Furthermore it may be a question whether secondary infection is of the saprophytic or the true suppurative type. In other words are the organisms virulent or nonvirulent? In the saprophytic type of infection the cytologic picture shows a mixture of eosinophiles and neutrophiles, the former somewhat predominating the latter. With true suppurative infection the neutrophile definitely predominates the cytologic picture with complete or almost complete exclu-

sion of the eosinophile. Since positive bacteriologic findings are the rule in these cases it is evident that a decision must be reached as to their significance. The cytologic picture is therefore a good indicator.

When this point in the diagnostic investigation has been reached should operative interference be instituted or should allergic management be tried first? With few exceptions we have adopted the policy of giving allergic methods a thorough trial before arriving at a decision as to what should be done surgically. Otherwise there is no method of determining in which cases there is marked improvement with recession of the polypoid or edematous changes or in which cases only slight or no improvement occurs. It is not unusual to note that the worst cases may show the most satisfactory improvement.

In the management of nasal polyps most otolaryngologists feel that they should be removed immediately. When nasal obstruction is complete the patient should have some immediate relief by partial or conservative removal of the polyps. In those instances in which the symptoms of sneezing and discharge are pronounced and the polyps are very edematous with little or no secondary fibrosis, conservative removal is advisable, for these are the cases in which recurrence rapidly occurs, within two to four weeks. On the other hand these are the type of polyps which show the most marked recesssion under adequate allergic management. The more marked the fibrosis the less the reduction, even with marked improvement in symptoms. As a rule recurrence is a much slower process in these cases.

In those cases in which nasal polyps are not removed and before allergic management has begun the degree of existing polyposis in the nose and in the paranasal sinuses should be recorded. The following plan of grading these changes is suggested.

Grade I. Nose: edematous thickening in middle meatus with no pedunculation. Sinuses: slight thickening of the mucosa.

Grade II. Nose: definite polyposis filling the middle meatus. Sinuses: moderate thickening of the mucosa.

Grade III. Nose: polyps extending down to the upper border of the inferior turbinate. Sinuses: marked edema or polyposis occupying about 75 per cent of the lumen.

Grade IV. Nose: complete obstruction with polyps. Sinuses: complete occlusion of the lumen.

Intermediate grades may be designated as one plus, two plus, etc.

In some cases there may be a difference in grade on one side as compared with the other. After the patient has been under treatment by allergic methods the degree of improvement may be noted by a change in the grade.

In the management of allergy of the nose and paranasal sinuses the most satisfactory results are dependent upon adequate allergic treatment. In some instances, however, some type of surgical interference is also necessary. Adequate allergic management is primarily dependent upon the skill of the allergist or the otolaryngologist who conducts his own allergic investigation and treatment.

With few exceptions the problem of allergic treatment concerns the consideration of such etiologic factors as the pollens, house and occupational dusts, and foods. Either one or a combination of these factors is involved. In the majority of cases of respiratory allergy the inhalants are the principal cause of symptoms, if no other manifestations such as gastrointestinal allergy, allergic headache, or skin allergy are present. If the latter manifestations are present, food must be considered a factor. In the absence of these symptoms food is not often a factor in the respiratory allergy.

Although good results may follow the avoidance of the causative inhalants, in the majority of cases injection therapy is necessary for satisfactory relief of the symptoms. In the injection of dust and pollen extracts the most important problem is the determination of the most effective dose. The universal principle adopted by most allergists today is to give the patient the largest dose he can take without aggravating the symptoms or producing constitutional reactions. By this method of treatment satisfactory relief is apparently the result of producing a state of disimmunity or refractoriness. Anything short of this is likely to aggravate the symptoms.

On the other hand good results may be obtained by the use of small doses rather than large ones, at the same time eliminating the danger of local and constitutional reactions. This method of treatment is almost entirely foolproof. The mechanism involved is apparently the result of producing a maximum of tolerance to the etiologic agent. In other words a maximum of protective substance is produced rather than a state of disimmunity or refractoriness. On the whole our results have been better by this method of treatment. In the administration of dust extracts we begin with a subcutaneous dosage of about 0.10 cc. of a 1:100,000 dilution at five to seven day intervals and increase it slowly until an effective amount has been reached. It is then maintained at this point and an attempt is made

to increase gradually the intervals between injections to ten, fourteen or twenty-one days. If the interval cannot be lengthened the dosage may be increased. Maximum effective doses usually reach from 0.10 cc. of 1:10,000 to 0.10 to 0.20 cc. of 1:1,000. Doses as strong as 1:100 or 1:10 are rarely used.

The same principle of subcutaneous dosage may be used in pollen therapy starting the average patient for example with 0.10 cc. of 1:1,000,000 ragweed extract. The dosage is gradually increased to 0.10 to 0.20 cc. of 1:10,000 and sometimes to 0.10 to 0.20 cc. of 1:1,000 as a maximum dose. We have not used larger doses. Local or constitutional reactions are extremely rare with this plan of treatment.

Hay fever may also be treated by the intracutaneous method using small doses of 0.03 to 0.05 cc. of that dilution which will produce a wheal about 18 to 20 mm. in diameter.

For further details on dust and pollen therapy the complete articles listed among the author's references below should be consulted.[72-73]

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Clinical Notes

XCVI

PENETRATING WOUND OF THE NECK WITH UNUSUAL COMPLICATIONS AND SEQUELAE*

HENRY BOYLAN ORTON, M.D.

NEWARK, N. J.

A white, 43-year-old man consulted his physician on Nov. 17, 1941, complaining of hemoptysis since Oct. 13, 1941. One week before onset, on Oct. 6, 1941, while working, he had accidentally run a wooden splinter into the right side of his neck. The patient pulled the splinter out at that time and thought nothing more of the incident. He went home to bed for a day and then returned to work. He had dysphagia for about a month following the accident, but this had disappeared.

The hemoptysis recurred about twice weekly, was small in amount, and was not associated with cough. The patient stated that the blood welled up into his mouth, and then he would expectorate it. There was no fever at any time. His general health had been good. Except for the indistinct scar where the splinter had entered the neck, nothing was found on physical examination. Fluoroscopic examination of the chest and esophagus was negative. Under laryngoscopic examination, fresh blood could be seen welling up into the trachea.

The patient was admitted to Fitkin Memorial Hospital on Dec. 10, 1941, with a provisional diagnosis of bronchogenic carcinoma, pulmonary tuberculosis, or foreign body. Rest in bed made no difference in the recurrence of hemoptysis. At this time there were some signs in the right base. X-rays showed a pneumonitis, with no evidence of neoplasm or tuberculosis, but two weeks later these physical and x-ray signs had entirely subsided.

*Presented before the American Laryngological Association, May 26, 1942, Atlantic City, N. J.

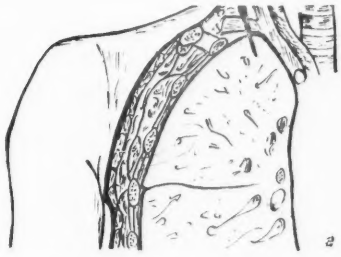

Fig. 1.—Schematic drawing of the foreign body, showing its relationship to the subclavian vein and apex of the lung.

I saw the patient on Jan. 3, 1942, at which time examination revealed a small, movable nodule in the supraclavicular fossa on the right side. The larynx was negative for paralysis.

A bronchoscopy at this time was negative for any ulcerations, stenosis, or tumors, and showed no trace of any bleeding.

On Feb. 17, 1942, his physician informed me that since the patient had left the hospital, he had had some hemoptysis and some glands had become palpable in the right axilla. The patient was admitted to the Presbyterian Hospital in Newark on Feb. 20, 1942. The urine was negative. The blood count showed: hemoglobin, 74; erythrocytes, 4,190,000; leukocytes, 13,800; color index, 0.9; neutrophiles, 46. After a rather severe hemoptysis his red count went down to 3,430,000, the hemoglobin to 64 per cent. The sedimentation rate was normal, and the sputum negative for tuberculosis.

It was necessary to give him five transfusions over a period of a month.

X-rays taken Feb. 21, 1942, the day after his admission, showed the bony framework to be normal and the apices of lung fields to be clear. There was no evidence of disease in the chest.

During the period of Feb. 20 to March 30, 1942, it was necessary to do a pneumothorax eight times to control bleeding.

On March 25, 1942, while being laryngoscopically prepared for bronchoscopy, the patient coughed up some blood. Bronchoscopy was done and the blood traced definitely to the right upper lobe bronchus.

Blood was taken for histological study and reported to be a "probable bronchogenic carcinoma." With the gland in the supraclavicular fossa and the pathologic diagnosis of carcinoma, preparations were made to remove the gland for biopsy. Palpation of the gland caused a fair amount of blood to be expectorated.

On April 4, 1942, the chest was opened in the third interspace and the ribs retracted. The upper lobe on the right side was adherent to the dome of the thoracic cavity, with firm infiltration around the apex of the lung and with what appeared on first palpation to be a calcareous substance on the under-surface or posterior surface of the upper lobe bronchus. The mere manipulation of this area caused the patient to have quite free bleeding through the mouth. In order to control the hemorrhage, a tourniquet was applied around the upper lobe. In removing the upper lobe we cut down onto a foreign body, which proved to be a splinter of wood, two inches in length. When the upper lobe was removed, we could see the splinter protruding into the thoracic cavity and in removing this, there was considerable hemorrhage. This was controlled, but it was necessary to transfuse immediately at the time of operation.

This splinter had penetrated through the neck into the chest, into the apex of the lung, and in so doing, it had either nicked the subclavian vein or had eroded it from pressure.

Infection followed the lobectomy and it was necessary to drain that area with rib resection.

At the present time, the patient is doing well, has a normal temperature, but there is still drainage from the chest. Had we removed the gland in the neck, I am sure we would have had uncontrollable bleeding in the lung.

224 DELAVAN AVE.

XCVII

ABSCESS OF THE PARAPHARYNGEAL SPACE: MASSIVE HEMORRHAGE, RECOVERY A CASE REPORT

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Milwaukee

Ligation of the common carotid artery or one of its branches should not be decided upon lightly, for this procedure itself, for whatever cause it may be done, carries with it certain inherent dangers and even death, and yet the hemorrhages which can and do occur in infections of the fascial spaces of the neck are so massive and terrifying that no other procedure can save the life of the patient. Particularly is this so in abscesses of the pharyngomaxillary or parapharyngeal space, for this space is in intimate connection with the great vessels of the neck.

The following case report concerns a boy, aged 11, who entered the Milwaukee Children's Hospital on February 12, 1938, with the history that six days previously, following a cold, he had developed a stiff neck. A myringotomy had been done for pain in the left ear with no relief. He did not appear acutely ill but he had enlarged, tender cervical glands on the left side. The neck was stiff. There was a small bleb on the left drum and a pharyngitis was present. A diagnosis of cervical adenitis and otitis media was made.

On February 15, 1938, a swelling of the left pharyngeal wall behind the posterior pillar was noted. There was trismus. This swelling increased and evidenced fluctuation. A diagnosis of abscess of the parapharyngeal space was made, and this abscess was evacuated through an incision along the anterior border of the sternomastoid muscle. Because of swollen, infected glands along its wall, a double ligature of the internal jugular vein was done. The pharyngomaxillary space was entered just above the hyoid attachment of the posterior belly of the digastric muscle and was explored by a finger in the external wound palpating against a finger in the mouth. A large amount of pus was encountered and drained. The wound was drained by a gauze drain. This operation was performed by Dr. Forrester Raine and myself. A transfusion of 350 cc. whole blood was given on the following day.

On February 21, at 11:05 A. M., the dressings were completely saturated with blood. The bleeding was apparently controlled by a large gauze pack in the wound and another blood transfusion was given.

On February 23, at 1:00 A. M., there was a large spontaneous hemorrhage from the external wound with the loss of from 300 to 400 cc. of blood. The gauze pack was removed and the bleeding was found to be coming from a vessel which was identified as the ascending pharyngeal artery. After considerable difficulty this was ligated.

The bleeding stopped but the boy continued to have pain in the left ear and to run a septic course. There was no mastoid tenderness and the x-ray films showed evidence of a breaking down of all the mastoid cells. A previous x-ray film, made several days before, had indicated some clouding and haziness in the posterior cells. On March 6, a simple mastoid operation was done. The boy continued to have a septic temperature until about March 11, after which he made an uneventful recovery. His throat culture, taken on February 13, had revealed a staphylococcus hemolyticus. Several blood cultures had all been negative.

Death from uncontrollable hemorrhage has occurred frequently enough after an intra-oral incision of a pharyngomaxillary abscess to impress upon one the necessity of using the external route. When such a catastrophy occurred, it was thought that an aberrant vessel or an aneurysm had been encountered. This may occasionally have been the case, but the true explanation in the majority of the cases lies rather in the fact that the vessels are slowly eroded by the necrotising, gangrenous effects engendered by the infection itself. This case report illustrated the fact that such erosion can take place days after the abscess has been drained. It also illustrated the fact that the hemorrhage was sufficiently controlled by packing until the offending vessel could be found and ligated. This would not have been possible through an intra-oral approach.

324 E. WISCONSIN AVE.

XCVIII

TRACHEAL ECTASIA

WITH REPORT OF A CASE*

ROBERT E. PRIEST, M.D.

MINNEAPOLIS

As is the case with many unusual conditions, there are a number of papers in the literature on tracheal ectasia. Most of them deal with one case because of the infrequency with which tracheal ectasia is encountered. This paper presents an additional case. The literature has been well reviewed by several authors,[2, 8] and only enough material will be cited here from other reports to form a background for the present case.

Tracheal ectasia is reported under various other names: tracheal hernia, tracheocele, tracheal diverticulum, aerial goiter, aerocele, and bronchocele. In the case reported here there was no proof that there actually was a sac attached to the trachea. The trachea was unquestionably dilated beyond normal limits. Therefore the name "tracheal ectasia," the term used by Choruzenko,[2] seems appropriate.

CASE REPORT

P. S. (U. H. 706403), a white male, in good general health was first examined in the nose and throat clinic of the University Hospitals, March 25, 1941. He was hoarse. His voice was high-pitched and sounded strained. He complained of pain in the supraclavicular nerve distribution bilaterally.

The man said he had been well until eight months before. He then became hoarse and began to cough. The hoarseness grew worse for five months, then stayed the same. About one year before he had noted a soft swelling on the right side of his neck just above his collar bone. This could be produced at will by closing his glottis and straining (Figs. 1, 2, and 3). He had slight dysphagia at the beginning of meals.

General examination, including urinalysis, blood studies, and serologic tests for syphilis, revealed no significant abnormalities.

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*From the Division of Otolaryngology, University of Minnesota Medical School.

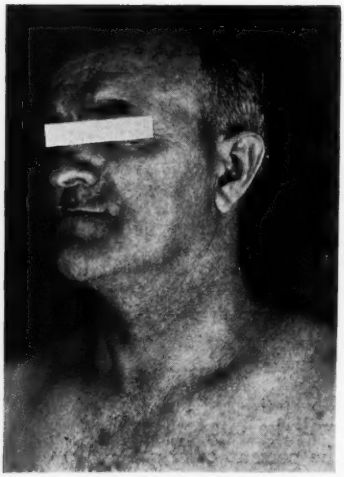
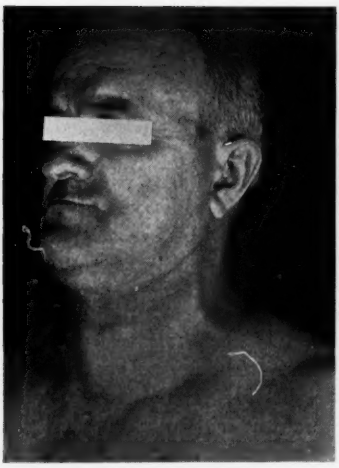


Fig. 1.—Showing the patient in normal quiet respiration.



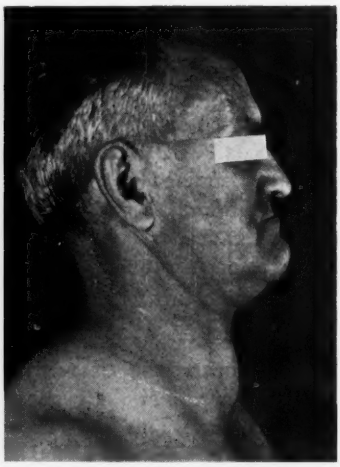


Fig. 2. Fig. 3.

Fig. 2.—Showing the distention of the neck on forced expiration against a closed glottis.

Fig. 3.—Showing the "aerial goiter" from the side during forced expiration with glottis closed.

Fig. 4.

Fig. 5.

Fig. 4.—Lateral roentgenograph showing the trachea and other airways in quiet respiration.

Fig. 5.—Lateral roentgenograph showing the tracheal dilatation on forced expiration. The triangular tumor mass is seen projecting forward from the ectatic posterior tracheal wall.

With a laryngeal mirror a papillomatous mass could be seen attached to the inferior surface of the left vocal cord. The cord moved normally. The larynx was entirely normal except for the papilloma. The cords nearly closed on attempts at phonation if the mass did not blow upward and become interposed between them.

A second wedge-shaped papillomatous tumor projected forward from the posterior wall of the trachea. It could be seen in the interarytenoid space on mirror laryngoscopy.

Roentgenologic examination included conventional and laminographic views of the larynx. The films made in the ordinary way were most informative. They are reproduced here. Fig. 4 shows the trachea during normal respiration. Fig. 5 demonstrates the dilatation occurring with forced expiration against a closed glottis. The wedge-shaped tumor can be seen projecting from the herniating posterior tracheal wall.

Fig. 6.

Fig. 7.

Fig. 6.—Drawing of the glottis as seen through the direct laryngoscope.

Fig. 7.—Drawing of bronchoscopic view of the tracheal tumor mass.

The chest film showed calcification of a portion of the left pleura. Fluoroscopic examination of the esophagus while barium was being swallowed demonstrated a normal esophagus.

Direct laryngoscopy was carried out on March 28, 1941. The larynx appeared as shown in Fig. 6. The papilloma was not removed until the trachea had been explored. This was done through a 7.0 mm. bronchoscope. A triangular roughened mass projected forward from the posterior tracheal wall at about the level of the seventh cervical vertebra (Figs. 5 and 7). The base was posterior and the apex anterior. This mass was removed until its broad, firmly attached base appeared to be clean. The tissue had the appearance and consistency of ordinary multiple laryngeal papillomatous tissue when seized with cup forceps. After the tracheal neoplasm was removed, the direct laryngoscope was reinserted and the tumor was excised from the left vocal cord.

Histologic sections were made from the tissue removed from the larynx (Fig. 8) and trachea (Fig. 9). The microscopic structure of the two was similar. A thick layer of stratified squamous epithelium was arranged to form a roughened, gnawed surface. The more superficial layers were formed by desquamating cells. A loose central stroma showed a moderate number of capillaries in the laryngeal papilloma, but a great many vessels in the tracheal specimen.

The patient has been well. He has returned twice for removal of papillomatous tissue from the larynx and trachea. The tracheal dilatation remains unchanged.

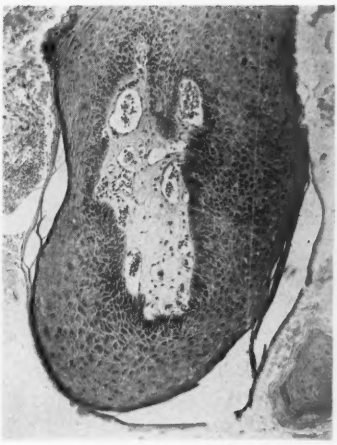


Fig. 8.

Fig. 9.

Fig. 8.—Photomicrograph of section from laryngeal papilloma. Sheets of squamous cells are seen overlying a moderately vascular stroma. There are no metastases or evidence of invasion by the deeper cells. There is surface keratinization and desquamation.

Fig. 9.—Photomicrograph of section from tracheal papilloma. This section is like that shown in Fig. 8 except for more vascular stroma.

DISCUSSION

As one watched this patient's neck enlarge when he inflated his aerial goiter, one got the impression that the right side ballooned out more than the left. If a tracheal diverticulum projected around the right side of the trachea with its opening on the posterior wall of the trachea, it should produce this appearance. There was no proof that a diverticulum existed here. The only evidence was a shadow marking the presence of air in the tissues at the side of the trachea (Fig. 5). This area of decreased density was separate from the main tracheal dilatation.

It is interesting to speculate on the reasons for tracheal herniation. The breakdown of tracheal mucous glands with herniation through the resulting defect has been postulated.[2, 8] A case report by Choruzenko[2] illustrates this theory. At autopsy in his case a sac was found communicating with the trachea laterally and behind. A caseous gland bordered on the stoma of the diverticulum.

The presence of amuscular areas between the bands of the trachealis muscle have been noted by Miller.[7] Such areas are thought to provide the weak points through which the tracheal mucosa bulges.[1] That the usual area for hernia formation is the posterior tracheal wall is agreed upon by most writers. Here the tracheal wall is formed by the trachealis muscle and cartilaginous support is absent.

Increased intratracheal pressure in the presence of a weak area in the tracheal wall is probably an important causative factor in tracheal hernia formation. The condition has been noted in minaret criers in Egypt, in drill sergeants, and following the excessive pressure caused by bursting of a large shell. A soldier was blown forty feet by a shell explosion and developed a tracheal hernia. Three rings of his trachea were found to be broken. A similar case is said to have followed the exertion incidental to moving a cannon.[8]

Petit[2, 8] studied 41 cases. He believed that a diverticulum may lie dormant between the trachea and esophagus until some expulsive force, such as persistent cough, forces it to open and to be distended.

Grenet and his coworkers[3] reported the case of a 3½-year-old boy before the Paris Pediatric Society in 1940. The child had a severe coughing fit. Afterward a soft mass formed on the side of his neck beside his trachea. The mass was resonant on percussion. It was aspirated and contained air with a little clear fluid. Laryngoscopic findings were normal. An attempt to fill the cavity with lipiodol was unsuccessful.

The number of sacs attached to any one trachea is variable. A patient treated at a U. S. Veterans' Bureau Hospital had a series of saccular dilatations along the posterior aspect of the trachea and proximal portions of the large bronchi. Some were "as deep as 7.0 mm." They were filled with lipiodol and the diagnosis was made ante mortem and confirmed post mortem.[4]

The finding of tracheal papillomata is interesting and unusual. Out of 314 patients treated for papillomata at Jefferson Hospital in Philadelphia from 1917 to 1936, there were 312 who had laryngeal papillomata and only three who had tracheal papillomata.[6]

A case of a tracheal diverticulum with papillomata about its tracheal orifice was reported by Kernan[5] in 1936. This sac ended at the innominate artery of a young infant. The party wall broke down resulting in fatal hemorrhage. Kernan's case is exactly similar to the one I am reporting in that papillomata were found in the

presence of tracheal ectasia. Is it not possible that the papillomata in the present case may have formed about the mouth of a tracheal diverticulum in response to irritation? Several points affirm this theory. The external mass was present for one year, but the cough was only noted eight months before the patient was first seen. Could not the cough have begun when the tracheal papilloma grew large enough to stimulate the tracheal sensory nerves?

The hoarseness was caused by a laryngeal papilloma younger than the tracheal tumor as shown by the large number of vessels and the fibrous stroma of the tracheal neoplasm. Therefore, the laryngeal papilloma may have been an implant from the tracheal one.

SUMMARY

A case of tracheal ectasia is presented. The possibility of tracheal diverticulum is considered. The unusual complication of tracheal papilloma is discussed.

1008 MEDICAL ARTS BLDG.

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XCIX

OTITIC MENINGITIS*

GUERDAN HARDY, M.D.

SAINT LOUIS

Within the space of a few years we have seen a definite drop in the mortality from otitic meningitis. Although the percentage of recoveries is still relatively small it is safe to predict that with the development of new drugs and with advances in the administration of drugs now at our disposal, there will be even fewer deaths. At the present time practically every patient who has recovered from otitic meningitis has done so as a result of the use of one of the sulfonamide drugs.

It is not the purpose of this communication to discuss the medical or surgical treatment of otitic meningitis in detail but rather to emphasize, with the aid of case reports, the importance of not relying solely on drug therapy. Regardless of the success of the medical treatment, the patient should not be discharged from the hospital until the suspected focus has been eradicated.

In the past few years there have been dramatic changes in the care of these patients. Prior to the advent of the sulfonamides the patient was taken to the operating room as soon as the diagnosis was made, providing that his condition permitted operation. In recent years, however, the medical treatment has been considered more important than the surgical care in the initial stages. The success of drug therapy has been so striking that in some instances it has been responsible for a false sense of security.

Needless to say, every effort should be made to determine the type of meningitis and, if possible, to establish the original site of entry or focus. The latter is not always easy. Assuming that epidemic meningitis has been ruled out, one should not suppose that, because there is an otorrhea, the focus must be present in the middle ear or the mastoid. The patient, or more often the family, generally gives a history of an upper respiratory infection preceding the onset

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Submitted as a Thesis for admission to the Ear, Nose and Throat Club of St. Louis.

of the meningitis. Hence, the nose, the nasopharynx, and the sinuses must be examined with thoroughness, and proper measures must be taken if infection is found.

Attention is directed to the ear by the history, the local evidence of otitis media or mastoiditis, and the laboratory examinations. Concerning the history, one should not be misled by a statement that the ear began to pain or to discharge only a day or two prior to the onset of the meningitis. While it is possible for a meningitis to occur with an acute otitis media, it is my impression that this rarely happens. If one is persistent and makes a determined effort to get the facts, he is usually rewarded with a history of otalgia or otorrhea, perhaps of short duration, some two, four or more weeks prior to the onset of the meningitis. The initial attack of otitis media may have been so mild that neither the patient or family sought the aid of a physician.

As soon as possible after the admission examination, mastoid and sinus x-rays should be made. Blood counts and cultures, and smears and cultures of the nose, throat, ears and spinal fluid are always indicated. At the time of the spinal puncture, manometer readings should be made before and after compressing the jugular veins.

The medical treatment should be started promptly and it must be heroic. The drug chosen for administration should be the one with the greatest bacterial coverage and with the best diffusibility into the spinal fluid. (Sulfathiazole does not enter the spinal fluid easily.) If the organism in the spinal fluid can be identified, the drug with the greatest inhibiting effect is chosen. Massive doses by mouth (or nasal tube), intravenously, subcutaneously, and intrathecally are indicated. Procedures, such as continuous spinal drainage and continuous administration of fluids parenterally, are generally necessary. If one waits to determine the benefits of a single type of treatment, he has usually waited too long. Other procedures (such as administration of sera and laminectomy) and the details of the medical treatment are not within the scope of this paper.

If the medical treatment has been successful in controlling the infection the next question is when should there be surgical intervention. This is more often than not a difficult problem and cannot be solved dogmatically. The patient's ability to survive the operation, the course of the infection, the blood picture, and many other factors must be taken into consideration. However, even though there is an apparent cure by drug therapy, the patient should not be discharged until the infected ear has been explored and a mastoidectomy performed. In my opinion, the mastoidectomy should be completed

even though the ear has ceased discharging and the ear drum is resolving. The decision as to whether this be a simple or a radical procedure must rest on the surgeon's judgment at the time of the operation.

For fear of being misinterpreted the following comment is made. Much has been written on the masking effect of the sulfonamides, and many otologists have condemned the use of such drugs unless one is dealing with an unusually severe infection or with the complications of mastoiditis. I am no adherent of such principles or policies but, on the other hand, advocate early and intelligent use of chemotherapy. This is not a discussion of when or how to use the sulfonamides in otitis media. It is to be noted that all of the cases reported had abnormal spinal fluid or a meningitis prior to the administration of the drug.

REPORT OF CASES

CASE 1.—E. H., aged 55, a white, married woman, was brought to Barnes Hospital Jan. 31, 1941. She had been irrational for the preceding twelve hours. Twelve days prior to admission she had had a head cold and an intermittent pain in the right ear. For a few days before admission there had been pain behind the right ear and before becoming irrational she complained of severe headache. Codeine and sulfathiazole had been prescribed by her local physician. Interrogation of the family revealed that the patient had complained of pain in, and behind, the right ear some two or three weeks prior to the onset of the present illness.

The patient was obviously acutely ill, stuporous, breathing rapidly, and the neck was rigid. The pulse rate was 138, the blood pressure 150/80, and the temperature 106 F. There were positive Kernig and Brudzinski signs (bilateral) and generalized hyperreflexia. There was dullness over the base of the right lung posteriorly and laterally, and diminished breath sounds and fine moist râles were heard over this area. The internist noted that the right ear canal was filled with debris, but no note was made about the condition of the ear drum.

Laboratory specimens taken at the time of admission demonstrated the following: RBC 4,970,000; WBC 19,450; NPN 28 mgm%. The blood culture remained sterile after five days. From the ear, nose, and throat, H. streptococci, H. staphylococcus aureus, staphylococcus albus, and pneumococci (unidentified), were grown. Pleomorphic gram positive organisms were found in a smear made from the spinal fluid but no bacteria could be demonstrated either by

culture or by mouse inoculation. The spinal fluid cell count was 2385 and the Pandy test was strongly positive. The initial pressure was 410 mm., with a rapid rise and fall following pressure over each jugular vein.

Massive doses of sulfapyridine were administered intravenously, subcutaneously, and by nasal tube. Continuous spinal drainage was instituted and fluids were given parenterally continuously.

A request to examine the ear was not received until Feb. 2, 1941, at which time the following items were noted: The right ear canal was filled with pulsating bloody fluid; the ear drum was dull, deep red, thick, and full. The fullness was especially marked superiorly and posteriorly, while in the lower posterior quadrant a small nipple-like projection was noted. No fluid escaped from this point. Mastoid and sinus x-rays were made and the reports were as follows:

Stereoscopic views of the petrous pyramids were negative. The mastoid films showed an increase in density about the canal on the right and the trabeculae were poorly defined, particularly in the cells about the periphery. The cellular structures on the left side were clear. The sinus film (Water's position) revealed a dense left antrum and a thickened membrane in the right..

The diagnoses were right otitis media and mastoiditis, bilateral sinusitis, and meningitis. It was recommended that a right mastoidectomy be performed as soon as the patient's condition permitted.

During the following nine days she improved somewhat on sulphonamide therapy. The mastoid x-rays were repeated on the ninth day and at this time a considerable increase in haziness over the cells of the right mastoid was noted. There was also a continuation of the breaking down of the trabeculae, and the groove for the lateral sinus was more prominent. With improvement in the clinical picture there was some indecision on the part of the internists as to whether or not the mastoid should be explored.

However, permission for operation was granted and a right mastoidectomy was performed on Feb. 14, 1941. Beneath the cortex were large necrotic cells leading to the mastoid antrum, in which a large amount of granulation tissue was found. Granulations were also noted on the wall of the lateral sinus near the jugular bulb. The dura appeared roughened, gray in color, and thickened. A stormy postoperative course ensued but she eventually recovered and was discharged from the hospital on May 30, 1941.

COMMENT

In view of the history of an upper respiratory infection and with the x-ray evidence of sinusitis, it probably would have been wise to irrigate the maxillary sinuses both for diagnostic reasons and in order to obtain a culture. There can be no question but that the massive doses of sulfapyridine kept this patient alive both before and following the mastoidectomy. As should be expected, there was destruction of the mastoid cells and granulations in the mastoid antrum and elsewhere, even though intensive chemotherapy has been instituted. Had she made a recovery without operation (which to my mind would have been temporary), a dangerous focus still would have remained within the mastoid.

CASE 2.—R. H., a white boy, aged 13, was readmitted to the St. Louis County Hospital Sept. 24, 1941. He had been discharged on Sept. 17, 1941, having apparently recovered from a pneumococcus (Type I) meningitis of otitic origin. Three days prior to the second admission he complained of a headache, had projectile vomiting, and experienced vertigo when walking. Subsequently the headache became more severe, his neck became stiff, he swayed to the right when he walked, and irritability developed.

The patient was in no apparent pain nor did he appear acutely ill. The head was held to the right and the neck was slightly stiff. Neurological examination revealed a left facial weakness, vertical nystagmus, weakness of the left sternomastoid, and positive Babinski and Oppenheim signs on the right. The patellar and abdominal reflexes and the fundi were normal. The admitting diagnoses were recurrent meningitis and brain abscess. Since he had previously improved with sulfapyridine, this was promptly administered by mouth and intravenously. Daily lumbar punctures were also instituted.

The otolaryngological examination on Sept. 26 revealed an intact, but retracted, left tympanic membrane, translucent but pinkish in color; diminished left hearing; tenderness over the tip of the left mastoid; peripheral left facial paralysis; horizontal nystagmus on looking to either side; and a suggestion of left external rectus paralysis. On douching the left ear canal with water at 56 F. an increase in nystagmus on looking to the right was produced but no vertigo was elicited. The lumbar puncture revealed no increased pressure or obstruction and the cell count of the spinal fluid was 65 with a preponderance of polymorphonuclear leukocytes. No organisms were grown from the spinal fluid.

X-rays of the mastoids showed haziness of the entire left mastoid structure with a loss of trabeculae. The diagnoses were left mastoiditis, left labyrinthitis, meningitis of otitic origin, and possible brain abscess. Exploration of the left mastoid was advised.

At operation small cells with thickened membrane and granulations were encountered throughout. The dura, which appeared injected, was uncovered extensively. A large portion of the lateral sinus was exposed and it seemed to be normal. No organisms were grown from mastoid cultures taken at the time of operation.

At first the postoperative course was satisfactory. Blood transfusions were given frequently, chemotherapy was continued, and the blood concentration of sulfapyridine was kept at a satisfactory level. Because of a kidney complication it was necessary on Oct. 3 to reduce somewhat the dosage of sulfapyridine and following this his condition became worse. Various neurological signs appeared and changed during the following days, producing a confused picture and making localization impossible. Administration of sulfadiazine did not alter the clinical picture and he expired on Oct. 30, 1941.

The autopsy revealed an acute leptomeningitis over the base of the brain with occlusion of the foramen of Monro and the foramen of Luschka, an acute suppurative ependymitis of the cerebral ventricles, and a slight internal hydrocephalus.

COMMENT

This patient was admitted to the hospital the first time during August, 1941, with a pneumococcus meningitis and left otorrhea. He was given sulfapyridine and, as the meningitis cleared, the ear ceased to discharge and the ear drum healed. At the time of the second admission there was no clinical evidence of an ear infection. The knowledge of the previous ear infection, the neurological signs and symptoms, and the destruction within the mastoid as shown by the x-ray directed attention to the left ear. In retrospect, a serious error was made in discharging this patient from the hospital without first performing a mastoidectomy, even though he was apparently cured of the meningitis and the ear infection.

CASE 3.—J. B., a white girl, age 6, was brought to the outpatient clinic of the St. Louis County Hospital on Nov. 1, 1941, because of an upper respiratory infection. She had a right serous otitis media which was treated conservatively and without chemotherapy. On Nov. 11, 1941, she was admitted to the hospital with a history of chills, fever, nausea, vomiting, general malaise, and joint

pains of 48 hours' duration. Soon after admission she complained of severe frontal headache.

The past history was significant in that a year before she had had scarlet fever followed by a draining ear (her mother did not recall which one) and a stiff neck. She had been seen by a physician only once at the onset of the scarletina; the otorrhea and neck rigidity had not been present at the time of his examination. In the months following she had episodes of unexplained nausea, vomiting, and vertigo lasting usually 24 to 48 hours.

At the time of admission her temperature was 104 F. The white cell count was 14,600 and the red count 4,650,000. Positive physical findings were large, inflamed tonsils, anterior cervical adenitis, and a slightly reddened, flat, dull right ear drum. An x-ray of the mastoids was reported as follows: "On the right side the mastoid is poorly aerated and the cells, especially in the antral region, are relatively opaque. There are no cells in the process. Condition on the right is probably due to an old mastoiditis."

Chills, a fluctuating temperature (99 to 105 F.) and headache continued without explanation. An x-ray of the accessory sinuses showed a peripheral opacity of both maxillary sinuses. So on Nov. 14 both antra were irrigated but the return was clear. The following day slight neck rigidity was noted, and upon performing a lumbar puncture the fluid was found to be cloudy and under increased pressure. The spinal fluid count was 1900, 75% of the cells being polymorphonuclear leukocytes; globulin positive; sugar 27 mgm%; total protein 129 mgm%. A smear revealed no organisms and the culture was sterile. A mouse was not inoculated at this time.

Positive neurological findings were neck rigidity, bilateral ankle clonus, positive bilateral toe signs, and positive Kernig signs. The fundi were normal. An examination of the ears, nose, and throat revealed nothing that could explain the origin of the meningitis.

With sulfapyridine the patient improved rapidly and apparently recovered. The mastoid x-ray was repeated Dec. 1, 1941, prior to discharge, and it showed less cloudiness than previously and there was no evidence of bone destruction.

For a week following discharge she was seemingly in good health but was readmitted on Dec. 10 because of the return of frontal headaches, nausea, and vomiting. Except for slight neck rigidity and a fever of 101 F., the physical examination was negative. Again the spinal fluid was cloudy and under pressure, the cell count being 3160

with 75% polymorphonuclear leukocytes. The smear was free of organisms and the culture was sterile. A mouse was inoculated with the spinal fluid and later examination of the peritoneal exudate showed gram-positive encapsulated diplococci.

The neurological report on Dec. 13 was negative except for a positive Babinski on the left and a biceps reflex greater on the left side than on the right. The eye grounds were normal.

My examination on Dec. 14 revealed no evidence of infection of the upper respiratory tract. Both ear drums were intact and flat. The whispered and spoken voice was heard at ten feet with each ear. The vestibular reactions were normal. An x-ray of the sinuses showed clearing of both maxillary sinuses. The mastoid x-ray, however, revealed an absence of cellular structure on the right and an opacity of the antral region.

Although there was no clinical evidence of middle ear or mastoid pathology, an exploration of the right mastoid was recommended on the basis of the history and the x-ray changes. At operation, Dec. 15, small cells with thick lining membrane were encountered and there was gross evidence of chronic infection in the tip and in the sinodural angle. The dura and the lateral sinus wall appeared normal.

Sulfapyridine was continued postoperatively and the improvement was gratifying. The spinal fluid on Dec. 27 was clear and under normal pressure; the cell count was 8 and all the cells were lymphocytes. Smear, culture, and mouse inoculation were negative. She was discharged from the hospital Jan. 25, 1942, and as far as is known has remained well.

COMMENT

The outcome of this case would probably have been unfavorable had the infection not been eradicated. Had it not been for the experience with the two preceding patients the operation might have been postponed until too late.

SUMMARY

Three cases of otitic meningitis are reported in detail; two of the patients recovered and one expired. Full credit is given to chemotherapy but attention is directed to the necessity of surgical removal of infected mastoid cells, even though there has been an apparent cure with drugs and definite clinical evidence of a mastoiditis is not present.

HUMBOLDT BUILDING.

C

A CASE OF VITAMIN DEFICIENCY WITH LARYNGEAL COMPLICATIONS*

ROBERT LINCOLN GOODALE, M.D.

BOSTON

This patient, a 66-year-old woman, was first seen at my office on Sept. 12, 1941, complaining of hoarseness and difficulty in swallowing.

Three months before this date, she first noticed some weakness of her voice. At times, this was severe enough to be called hoarseness. She also had some difficulty in swallowing, especially noted when she had to take pills, which seemed to catch on the left side of the throat. Some years previously, she had been treated for "acid indigestion" at another clinic. During the past three months, she had had several attacks of sore tongue, and puffiness around the lips. Occasionally, she had had painful areas in the palms of both hands and soles of both feet which, she said, seemed to burn. She also had had some difficulty in opening her eyes because of puffiness of the lids.

On examination, the tongue was somewhat smooth along the margins, but deeply furrowed in the midline. There were many teeth gone, and what remained suggested that there might be improper mastication. The posterior pharyngeal wall was redder and smoother than usual. The nose, the nasopharynx and the rest of the pharynx were negative.

On palpation of the neck, no swelling and no point of local tenderness could be made out.

Laryngeal examination showed a swelling involving the left arytenoid, the false cord, and the aryepiglottic fold. The arytenoid was slightly reddened also. The true cord was motionless in the midline position, but otherwise normal in appearance.

The patient was admitted to the Baker Memorial of the Massachusetts General Hospital for study. The Hinton test was negative,

*Presented before the meeting of the American Laryngological Association, Atlantic City, N. J., May 26, 1942.

as were also the renal function, sedimentation rate, and the red and white blood cells and hemoglobin. The basal metabolic rate was +2. X-ray films of the chest showed the lung fields to be clear, the diaphragm and heart shadow normal. The aorta was somewhat tortuous. Fluoroscopic examination of the esophagus showed:

"The patient holds the barium in the mouth without difficulty, and in the first portion of the act of swallowing, uses the tongue and pharyngeal muscles. The esophagus remains open as long as the patient is in the upright position, and the barium flows through without peristaltic action. In the horizontal position, there is some peristalsis in the upper third, but it is weak and there is definite narrowing and rigidity of the esophagus for a distance of about six centimeters above the aortic arch. Also, while the patient is in the horizontal position, the barium remains in the esophagus for a long period of time and there are no peristaltic waves seen in the lower portion."

On Sept. 26, laminography of the larynx showed a slight deviation of the larynx and trachea to the left. No definite defects were seen.

On Sept. 29, the gastrointestinal tract was examined under the fluoroscope with the following report:

"Further examination of the esophagus does not add to the previous findings. The stomach is low in position. It is smooth in outline and without defects. The duodenal cap and loop appeared normal. At one hour, there is only a trace of barium remaining in the stomach, and the head of the meal had reached the mid-ileum. At two hours, most of the jejunum and ileum are filled and at three hours, the head of the meal has reached the hepatic flexure. No abnormal loops of small bowel were demonstrated. No evidence of vitamin deficiency from the appearance of the small bowel is seen."

Esophagoscopy was performed under ether anesthesia. The report is as follows:

"The larynx was exposed with the Mosher laryngoscope. There was a smooth, edematous-like swelling of the left false cord, aryepiglottic fold and arytenoid. The pyriform sinuses were normal. The motion of the cords was hard to determine as the patient was in spasm most of the time. There appeared, however, to be no motion in the left cord, which agreed with the first examination at the office. The esophagus was entered easily and appeared to be patent throughout. No obstruction could be found. The mucous

membrane appeared to be smoother than normal. The esophagus collapsed on expiration but seemed to be more open and tubular than normal."

A biopsy was taken from the left arytenoid. It was reported to be chronic edema. There was no evidence of malignancy.

During the patient's stay in the hospital, the medical consultant brought up the possibility that this might be a case of scleroderma. The differential diagnoses which were suggested included also: healed mediastinitis, myesthenia gravis, bulbar palsy, and perichondritis of the larynx. The fluoroscopic examination of the esophagus so strongly suggested scleroderma that other studies of the gastrointestinal tract were carried out. These, however, failed to confirm this diagnosis.

Neurological examination ruled out both bulbar palsy and myesthenia gravis. Perichondritis was ruled out by the absence of local tenderness in the neck, the absence of painful sensation in the larynx, and by the character of the swelling which showed no evidence of inflammation. There was not sufficient evidence of healed mediastinitis so that, up to this point, the diagnosis was very much in doubt.

On going back over her history, it appeared that because of very bad dental equipment, she had been taking soft food only, and had definitely avoided meat, vegetables, and whole grain cereals. It was suggested that this might be a borderline vitamin deficiency case, because of the dietary story, the appearance of the tongue and the mucous membrane of the mouth. Consequently, she was given a preparation of vitamin B complex in capsule. The daily dose contained thiamin chloride, 6 mg.; nicotinic acid, 12 mg.; and 600 Bourquin Sherman units of riboflavin.

As she had been avoiding citrus fruits because of her old history of "acid indigestion," she was told to take three glasses of citrus fruit juice daily. She was then discharged from the hospital on Oct. 6.

On Oct. 20, she reported at my office. She then said that there was an almost immediate improvement in the sensations of burning which she had had in her hands and feet. In fact, these have entirely disappeared. There has been no further puffiness of her eyelids and mouth.

On examination, the swelling in the larynx had begun to subside. This was first noted on the posterior surface of the arytenoid.

The left true cord began to show a little motion. Her vitamin intake was increased by two more capsules daily. On Nov. 3, the swelling had still further subsided, but was still present in the ventricular region. However, there was no difficulty in swallowing. There was a little fatigue in her voice at the end of the day. Two weeks later, the vocal cord was moving more normally, and a month later, there was even greater improvement.

On March 16, 1942, the recovery was complete. X-ray of the esophagus was repeated on March 19 for a check-up, and the report is as follows:

"The esophagus shows a considerably different appearance than at the previous examination. At this time, there appears to be some spasm in the lower portion of the esophagus, similar to that seen in cardiospasm or idiopathic spasm of the lower portion of the esophagus. The upper two-thirds of the esophagus appears to remain open and contains considerable air when the patient is in the upright position. Muscular contraction is poor throughout the esophagus, and it is somewhat dilated. In the horizontal position, the patient has a great deal of difficulty in emptying the esophagus. From the present examination, it would seem more likely that this represents a form of spasm of the lower end of the esophagus, which is probably of mild degree but of considerable duration. It would seem likely that when we saw her at the previous examination, there was no spasm and the barium simply dropped through the esophagus when she was in the upright position; and when in the horizontal position, the flabby muscular walls could not contract sufficiently to force the barium into the stomach."

It would seem that this case was one of intermittent obstruction of the lower portion of the esophagus which, in the course of many years, had led to dilation of the esophagus and some weakening of the muscular walls. Superimposed on this, was a dietary problem due to poor chewing which led to a choice of food deficient in the vitamin B complex. The disturbance of the mucous membrane of the mouth and pharynx suggests a lack of the nicotinic and riboflavin elements. The painful areas in the hands and feet, and possibly the loss of motion in the left vocal cord, are suggestive of a lack of antineuritic vitamin B_1.

The case is presented to call attention to the possibility of laryngeal manifestations of vitamin deficiency.

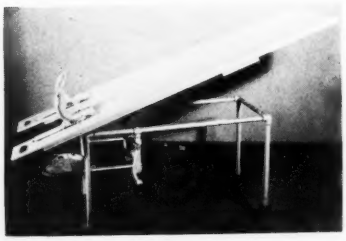
330 DARTMOUTH ST.

The Scientific Papers of the American Broncho-Esophagological Association

(Continued from the September Number)

CI

PRESENTATION OF SPECIMENS AND NEW INSTRUMENTS



A POSTURAL DRAINAGE BOARD

F. W. Davison, M.D.

Danville, Pa.

Postural drainage is a valuable means of assisting a patient to rid his bronchi of retained exudate. Because the procedure is fatiguing it is often not prescribed for the weak, debilitated patient who needs it most.

The adjustable board shown in Fig. 1 permits effective postural drainage with a minimum of fatigue.

The patient is placed on the board while it is horizontal. The top is then elevated to the desired angle by turning the crank which activates an ordinary automobile jack.

This board has been especially helpful in caring for patients with acute suppurative pneumonitis surrounding lower lobe bronchiectatic areas.

AN IMPROVED ELECTRIC LARYNGEAL SAW

MURDOCK EQUEN, M.D.

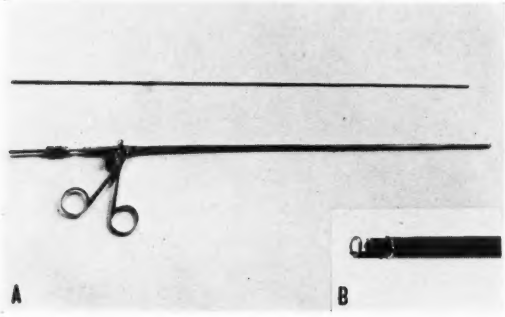
ATLANTA

Those of us who do bronchoscopy have watched the development of various instruments with a great deal of interest. The use of this electric saw, we feel, makes the division of the thyroid cartilage much simpler.

Some of us who have used an electric saw have experienced the embarrassment of having the saw suddenly get beyond our control and either to injure the surrounding tissue or become enmeshed in the nearby gauze and linen. With this instrument the depth of the saw can be gauged by twisting the screw.

This saw is of particular value in cutting through the thyroid cartilage for anterior commissure growths when it is desirable to go through only the cartilage leaving the mucous membrane intact.

The blade of the saw is removable and new blades may be inserted as desired.



AN INSTRUMENT FOR SEVERING STAPLES AND SIMILAR OBJECTS IN THE AIR AND FOOD PASSAGES

WILLIAM A. HUDSON, M.D.

DETROIT

The instrument consists of a tubular shaft, in the wall of which are two small canals which run the full length of the shaft. Through these small canals are threaded the two ends of a looped wire, the loop falling directly over, and bent at right angles to, the distal end of the main canal. The proximal ends of the wire are attached to the two jaws of a handle. Through the main canal is inserted a drill. This drill can be run by hand power or by an electric motor. It is the means by which the object is severed. The object is caught between the loop and the end of the shaft and is held in place by the loop, while the friction of the rapidly spinning drill causes the object to be severed. A set screw on the drill shaft prevents the drill from passing beyond the object into the adjacent soft tissues.

CII

FOREIGN BODIES IN THE GASTROINTESTINAL TRACT*

EMILY L. VAN LOON, M.D.

AND

SIDNEY DIAMOND, M.D.

PHILADELPHIA

When a patient with a swallowed foreign body which has become lodged in the esophagus presents himself for treatment, the problem confronting the physician is fundamentally that of the precise technic of endoscopic removal of the object from the patient's food passage; peroral removal is indicated in every instance. With the entrance of the swallowed foreign body into the gastrointestinal tract, however, the problem becomes more complex and its solution depends upon a variety of factors. In certain cases peroral endoscopy is the method of choice. In others the object may be removed by laparotomy. In a large number conservative watchful waiting, in anticipation of spontaneous passage of the foreign body by bowel, is the wisest policy.

Seeking to learn whether definite criteria might be established for the adoption of each of the aforementioned procedures in the management of cases of foreign body in the gastrointestinal tract, we studied the records of 386 consecutive patients seen in the bronchoscopic clinic at Temple University Hospital. In each case the swallowed object was radiopaque and was present in the stomach (38 per cent) or in the intestines (62 per cent) at the time the patient was first seen. The most common foreign bodies, in the order of frequency, were as follows: straight pins, 49; coins, 40; open safety pins, 40; closed safety pins, 29; bobby pins, 29; and nails, 26.

The greater number of the patients in the series were small children. The youngest was a baby 7 weeks old who swallowed an open safety pin and passed it in the stool two days later. The oldest was a 70-year-old woman who swallowed a denture. Of the 40 patients who had swallowed open safety pins, 19 (48 per cent) were infants less than 1 year of age. Of the 386 patients with foreign bodies in the gastrointestinal tract, 28 were less than 1 year old; 177 were be-

*From the Chevalier Jackson Bronchoscopic Clinic, Temple University Hospital.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 1.—Nail 6 cm. long in the intestine of a 2-year-old child. It was passed uneventfully.

Fig. 2.—Barette 4.6 cm. long in the intestine of 2½-year-old child. It appeared in the stool four days after being swallowed.

Fig. 3.—Hairpin 6 cm. long in descending portion of duodenum in 2-year-old child. It remained fourteen days, unable to complete the turns of the duodenum, but did not perforate duodenal wall. It was removed by laparotomy.

Fig. 4.—Open safety pin perforating wall of stomach in 8-month-old baby. The child's behavior gave no suggestion that pain or any other abnormal symptom was present at any time. The pin was removed by laparotomy.

tween 1 and 4 years of age; 66 between 5 and 9 years; 45 between 10 and 14 years; and 68 were older than 15 years.

There was no mortality in the group of 386 patients. Except for the original episode of choking and gagging, symptoms were present in only two patients. Endoscopic or surgical intervention was required in but 5 per cent of the cases, spontaneous evacuation of the foreign body occurring in the remainder. All blunt foreign bodies which advanced beyond the stomach were passed by bowel. Similarly, all straight pins, bobby pins, nails, tacks, picture buttons, toy glass eyes, dentures and needles reaching the intestine passed uneventfully. Patients from whom foreign bodies were removed by gastroscopy ranged in age from 9 months to 5 years. The foreign bodies included a part of a buckle, open and closed safety pins, bobby pins, a lead skirt weight, picture buttons, and a toy wrist watch. At removal they had been in the stomach for periods varying from less than one day to thirty-five days.

In 17 patients the foreign body did not pass beyond the pylorus. Removal was accomplished by fluoroscopically guided open-tube gastroscopy in 14. External operation was required in three. Of the endoscopically removed objects, seven were in the stomach only five days or less and their spontaneous passage would perhaps have occurred if intervention had been postponed. While it is true that the vast majority of the 369 foreign bodies which eventually found their way into the intestinal tract did so less than two days after being swallowed, some did so only after a comparatively lengthy period in the stomach. In one patient, a 50-year-old woman, a small locket remained in the stomach a full month, then passed through the pylorus into the duodenum and appeared in the stool two days later. The failure of the other seven foreign bodies removed by gastroscopy to pass through the pyloric opening is difficult to explain. Perhaps the pyloric orifice was abnormally small in these seven patients. Neither the excessively large size of the foreign body nor the youth of the child alone can be held responsible, for objects of equal and even greater dimensions passed without undue delay in children of similar ages.

Abdominal section for extraction of the foreign body was necessary in seven patients, gastrotomy being performed in three and duodenotomy in four. Failure of the foreign body because of its length and shape to complete the turns of the duodenum provided the indication for laparotomy in two of the patients. In both cases the object remained in the stomach but a few hours and was already in the duodenum at the time of the patient's initial visit to the

Fig. 5.

Fig. 6.

Fig. 5.—Two needles perforating the wall of the stomach in a 17-year-old girl. The film was made eight hours after ingestion of the needles. Perforation was accompanied by moderately severe epigastric pain. The needles were removed by laparotomy.

Fig. 6.—Large denture impacted in the terminal ileum of a 59-year-old man. It remained in situ six days, producing partial intestinal obstruction, and was finally passed spontaneously.

clinic. In one case the progress of the hairpin was arrested in the second portion of the duodenum, and in the second case a bent wire was arrested in the fourth portion. Penetration of the gastric or duodenal wall by the point of the foreign body led to laparotomy in the other five patients. The diagnosis of perforation was made by roentgenoscopy in each instance, being suggested by the persistence of the foreign body at a particular site along the gastrointestinal tract, despite attempts at manual or postural dislodgment during fluoroscopy, and being corroborated by the actual demonstration of mural penetration with the use of air or barium as a contrast medium to fill the gastric or intestinal lumen. In two of the patients in whom gastrotomy was necessary, preliminary gastroscopic efforts to free the intruder from its attachment to the gastric wall were unsuccessful. It is significant that in only one of the seven patients was perforation accompanied by symptoms: In the case of the 17-year-old girl who swallowed two needles, moderately

severe epigastric pain developed eight hours after the ingestion of the foreign bodies; roentgenographic examination with a barium meal revealed that both needles had penetrated the wall of the stomach.

In addition to those needing surgical intervention, there were seven patients in whom the foreign body because of its size or contour was temporarily delayed in its forward movement in the intestine. In six the swallowed object was a bobby pin which remained in the duodenum for from two to ten days before passing onward; the second portion of the duodenum was the point of lodgment in two, the third portion in two and the fourth portion in two. All six were children, ranging in age from 2 to 6½ years. It is interesting to note that the longest delays occurred in the two oldest children in the group: six days in the 6½-year-old child and ten days in the 4-year-old child. The seventh case of temporarily arrested progress of the foreign body in the intestine was that of a 59-year-old man who swallowed an artificial denture containing four lower incisor teeth and having the usual sharp hook at each end. The outside curve of the denture measured 7.2 cm. and the distance between the hooks was 5.4 cm. After an uneventful passage through the upper part of the alimentary canal, the denture became impacted in the terminal ileum. Partial intestinal obstruction, characterized symptomatically by abdominal pain, vomiting and constipation ensued. The obstruction gradually became more pronounced, and roentgenograms of the abdomen on the sixth day of impaction disclosed the loops of bowel proximal to the foreign body to be markedly distended with gas, with a number of fluid levels present. Operative intervention seemed inevitable, but at this juncture the obstructing denture suddenly moved along through the ileocecal valve and the following day it appeared in the stool.

CONCLUSIONS

From the data in our series of 386 cases it is obvious that the great majority of foreign bodies in the gastrointestinal tract do not have to be removed endoscopically or surgically. More than 90 per cent will pass spontaneously, even in very young children, although the time necessary for them to pass is sometimes unaccountably prolonged. A conservative policy of watchful waiting should therefore be adopted in most instances. Cathartics or any change in the patient's usual diet is definitely contraindicated. Early gastroscopic removal of pointed objects, under biplane fluoroscopic guidance, will of course obviate the danger of perforation and the necessity for an extended period of observation. But, unfortunately, in many cases

the foreign body has already advanced beyond the range of accessibility to the rigid gastroscope by the time the patient presents himself to the physician. In such cases the patient should be observed closely and the progress of the swallowed object followed until the foreign body appears in the stool. Patients who have swallowed a sharp object should be fluoroscoped daily to determine the exact position of the foreign body, particularly its relationship to the lumen of the alimentary tract. If perforation of the gastric or intestinal wall has occurred, as suggested by immobility of the foreign body upon manual manipulation and changes in posture, and as confirmed by delineation of the organ in question either with air insufflated through a rubber tube or gastroscope or with barium administered by mouth, immediate laparotomy is in order. It must be remembered that gastrointestinal foreign bodies produce symptoms only very rarely. If the diagnosis of perforation is to be made early enough to insure the performance of laparotomy before additional complications arise, it must be made by roentgenoscopy and not based on the patient's symptoms or physical signs.

1930 CHESTNUT ST.

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CIII

TWO CASES OF MEDIASTINITIS FOLLOWING ESOPHAGEAL FOREIGN BODIES

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Four days before admission, A. D. (Fbdy Case 190), a white, male plasterer, aged 46 years, had swallowed a chicken bone and had undergone two esophagoscopies in a neighboring city during the interim. His physician stated that he thought the foreign body had perforated the esophageal wall. The patient had moderate dysphagia for liquids but was not apparently acutely ill. X-ray films showed swelling of the prevertebral soft tissues from the level of the hyoid downward and a thin bone about one and one-half inches in length in the vertical plane between the cricoid and the cervical vertebrae.

Esophagoscopy showed swelling of the mucosa of the upper esophagus, a small vertical laceration in the left lateral wall just below the cricopharyngeus muscle and no other evidence of foreign body. Later that day a cervical mediastinotomy was performed with exposure of the esophagus from the level of the suprasternal notch to the level of the hyoid. The foreign body could not be found and no gross pus was encountered. The wound was packed with iodoform gauze.

The patient's progress was very unsatisfactory. He was unable to swallow even liquids and expectorated considerable purulent material. Two days after admission the esophagus was reëxamined by simultaneous esophagoscopy and exploration of the external wound which showed foul pus. A small slit-like opening was found about one and one-half inches below the previously observed laceration. As the foreign body could not be found it was thought that it had possibly reëntered the esophagus through this more recent laceration.

For the next ten days the patient was fed through a nasal feeding tube. He expectorated large amounts of pus and there was profuse foul purulent drainage through the tubes placed in the neck wound. By the tenth day he was able to take regular hospital diet readily. The wound in the neck continued to drain for four months

but finally healed and his physician reported that he was apparently perfectly well.

J. S. (Fbdy Case 214), a white male farmer, 48 years of age, had choked upon a bone while eating rabbit three days before admission. Swallowing had become increasingly difficult and painful and he had developed swelling and tenderness on the left side of the neck. X-ray films showed marked swelling of the retroesophageal and retropharyngeal soft tissues.

Esophagoscopy revealed marked swelling of the entire posterior wall of the pharynx and hypopharynx and of the entire mucosal lining of the esophagus. No laceration of the mucosa and no foreign body were found. Mediastinotomy was advised but was delayed because the patient and his family would not consent to surgery.

Twenty-four hours after admission there was marked swelling on the left side of the neck from the clavicle to the hyoid and on the right side of the neck from the ear down to the level of the hyoid. The patient had a high temperature, was slightly irrational and showed rapidly increasing laryngeal obstruction because of swelling in the hypopharynx.

An emergency tracheotomy was done with only partial relief of dyspnea. A small amount of foul pus was encountered in the tracheotomy wound. A left cervical mediastinotomy was then performed and dissection carried back to the vertebral column. The tissues were markedly edematous, the picture being that of a diffuse cellulitis rather than that of an abscess. No foreign body was encountered. Drainage of the retropharyngeal space was performed through an incision on the right side of the neck behind the angle of the jaw. Large drainage tubes were placed in the neck and the tracheal cannula packed off with bichloride gauze in a vain effort to prevent spillage into the trachea. A nasal feeding tube was also inserted.

During the succeeding week the patient was kept alive only by the most strenuous nursing care and vigorous supportive therapy, including administration of sulfonamide drugs. The drainage tubes were irrigated with Dakin's solution with evacuation of large amounts of foul pus and particles of necrotic tissue. Solution instilled into the wound in the right upper neck would drain from the tube in the left lower neck. The tracheotomy wound was necrotic. There were signs of pneumonitis in the right lung for four or five days.

However, the patient gradually improved. The tracheal cannula was removed and the wound plugged with vaseline gauze on the tenth postoperative day. The feeding tube was removed on the fourteenth day.

The neck wounds continued to drain for eight weeks. When seen five months later he was apparently perfectly well and the throat and larynx were normal to mirror examination.

604 Medical Arts Bldg.

CIV

THE TREATMENT OF LYE INGESTION BY THE SALZER METHOD

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AND

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The ingestion of lye is the most frequent cause of esophageal stricture during childhood. The availability and the widespread use of this material account for the frequency of poisoning, especially in small children, despite the publicity which has been given regarding the dangers of its ingestion. Fewer patients cause greater economic drain on a hospital than children with esophageal strictures resulting from lye burns.

Until 1924 it had been the policy at the Harriet Lane Home, of the Johns Hopkins Hospital, to treat the visible burns due to the lye and to neutralize the alkali with a weak acid. Strictures were treated only if and when they had developed. This method is still commonly used in the majority of hospitals in this country. In 1920 Salzer[1] published a brief article discussing the value of early treatment, in the hope of preventing the development of a stricture following the ingestion of lye, as opposed to treatment instituted after the stricture had already developed. He reported successful results in 12 patients in whom he started dilatations on the second to the sixth day after the ingestion of the caustic. In 1924 Bokay[2] drew attention to Salzer's paper and reported results in 132 patients in whom early dilatations were performed. He stated that his results in 117 of the patients were excellent.

In 1939 Martin and Arena,[3] in this country, reviewed the subject of lye poisoning and reported three cases successfully treated with early dilatation. Since 1924 the method outlined by Salzer has been routinely employed at the Harriet Lane Home. The great majority of the children seen in the dispensary because of lye ingestion have been treated as ambulatory patients, only the very badly burned children having been admitted to the wards.

The following is an outline of the treatment used at the Harriet Lane Home:

1. An attempt is made immediately to neutralize the corrosive with weak acid.

2. Visible burns are treated with an emollient and the child given a sedative to make further manipulation easier for the patient and doctor.

3. Dilatations are started with bougies in the form of soft rubber catheters, tapered and closed at the lower end, filled with small lead shot and corked above. These range in size from No. 14 to No. 34 French and are of sufficient weight and flexibility to descend the normal esophagus with little effort on the part of the operator. The bougies are passed, beginning with a small size and increasing the size used until No. 28 or No. 30 is reached or until difficulty is experienced in getting the bougie down. They are not forced, and should bleeding of consequence occur, the treatment is discontinued for the day. Unless there is great swelling of the fauces the dilatation is begun at once. It is not, in any event, postponed more than four days. The patient is always held in a sitting position during dilatation.

4. The amount of treatment required varies in each case. The dilatations are done daily for two weeks, three times a week for two weeks, twice a week for two months, once a month for six months and two or three times a year for several years. If increasing difficulty is encountered during the course of treatment, daily dilatations are again performed. Dilatation is begun each time with a bougie two sizes smaller than the largest used in the preceding treatment. It is well to have the largest bougie used in place thirty minutes if possible.

5. Fluoroscopy with barium should be done in all cases, preferably several times during the course of the treatment.

6. Esophagoscopy should be done in all cases if possible, especially if any difficulty is encountered in passing the bougie, if dysphagia or pain develops, if there are signs of a fistula or if much bleeding occurs.

7. Every patient is referred immediately to the social service department through which arrangements are made to facilitate the regularity of clinic attendance. On occasion the aid of the juvenile court has been necessary to enforce attendance.

RESULTS

A total of 110 children have been seen at the Harriet Lane Home with histories of lye ingestion. Twenty-eight of these children were white, 82 colored. Of these, 71 children were seen within a few hours following ingestion of lye, while 39 were first seen because of fully developed strictures.

Of the 71 patients seen shortly after lye ingestion, 41 were treated according to the routine outlined above. Fourteen children received no treatment but were adequately followed. The remaining 16 children received dilatations but either left the city or failed to return and so could not be adequately followed concerning the possibility of the development of a stricture.

Of the 41 children treated according to Salzer's method, only one developed a stricture. All of these patients have been followed at least a year. Of the 14 children who received no dilatations, six developed strictures.

During the course of the dilatations not a single accident occurred and there have been no deaths in this group. One child died during the period of acute symptoms before dilatations were begun.

In the group of 39 children who when first seen had fully developed strictures, there were eight deaths: four died following esophagoscopy as a result of rupture of the esophagus, one of an esophagotracheal fistula, one of peritonitis following gastrostomy, one suddenly following gastrostomy and one of pneumonia.

In summary, 43 per cent of the children in our series who were not treated developed strictures, while only 2.5 per cent of our patients treated according to the Salzer régime were found to develop strictures. These figures confirm the findings of Bokay.

HARRIET LANE HOME
JOHNS HOPKINS HOSPITAL

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CV

MYOBLASTOMA OF THE LARYNX*

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CINCINNATI

In 1926 Abrikossoff[1] described a form of tumor which he regarded as new and which he designated as myoblastenmyome. In all of his five cases the tumors had originated within muscular structures, three in the tongue, one in the upper lip and one in the gastrocnemius muscle. Abrikossoff, at this time, regarded these tumors as a degenerative process following trauma or inflammation in striated muscle.

Following this report Klinge[2] observed some tumors of similar structure, some of which, however, had arisen from tissues in which there was no striated muscle. This led Abrikossoff,[3] who, in the meantime, had observed some additional cases, to modify his original concept, and in his second report in 1931 he stated that some of the myoblastomas might originate from cell inclusions from the primitive myoblast of the embryo. This seemed to be confirmed by the microscopic structure of the tumors which consisted of two types of cells: first, very large elongated, finely granular cells arranged in bands and resembling the primitive myoblasts; and second, large polygonal or ovoid cells arranged in groups surrounded by fine strands of connective tissue.

By 1934, Klemperer[4] was able to collect reports on a total of 50 cases of myoblastoma. These tumors had been removed from various parts of the body, but, by far, the greater number had occurred in the upper digestive and upper respiratory passages. About three-fourths of them had been found in the mouth and pharynx, with a great predilection for the tongue.

In four of the cases cited by Klemperer the myoblastomas had originated in the larynx, and he reported an additional case which Kleinfeld[5] had submitted for microscopic diagnosis.

Since that time, three additional cases have been reported, one by Geschelin,[6] one by Bobbio[7] and one by Frenckner.[8] In seven of

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the eight recorded cases the tumor occurred as a solitary mass attached to one of the vocal cords. In the eighth case (Frenckner) the tumor was apparently encapsuled in the left aryepiglottic fold and left arytenoid region.

As a rule, these tumors did not exceed the size of a pea or bean and varied from a grayish white to slightly red color.*

In addition to these myoblastomas, true striated muscle tumors, rhabdomyomas, have been removed from the larynx by Imperatori[10] and by Kernan and Cracovaner[11] in one instance each, and by the Jacksons[12] in three cases, but these tumors belong in a different category. There also have been two cases of leiomyoma of the larynx mentioned in the literature.[11, 13]

The following case recently came under my care:

REPORT OF CASE

Mrs. G. W., a colored female, aged 41 years, was referred to me on Jan. 17, 1942. Her chief complaint was hoarseness which had been present for about one and one-half years. Three years before she had had a fibromyoma of the uterus removed, but otherwise her past history was unimportant.

Her present illness had begun about one and one-half years before when she had noticed that she was becoming hoarse, but there had been no increase in the hoarseness during recent months. There had been no choking sensation, pain, dysphagia, hemoptysis or dyspnea. She found it necessary to clear her throat, and snored in her sleep, but not more than in previous years.

Mirror examination showed a large pedunculated tumor in the posterior portion of the larynx between the arytenoid cartilages. The tumor was globular in shape and was the size of a hazelnut. Its surface was slightly corrugated and it was the color of butter. The mass moved upward during phonation, exposing the vocal cords which were normal in appearance and in motion.

One week later, under local anesthesia, the tumor was removed under direct laryngoscopy. The patient was very restless and uncooperative so that it became necessary to remove the mass in sec-

*Kramer[9] has reported an interesting case of myoblastoma of the bronchus and Frenckner[8] one in the trachea.

tions with a punch forceps. There was considerable bleeding during the operation.

The tissue was examined by Dr. P. Wasserman, who reported as follows: "Tissue from region of larynx, three sections: All show essentially the same picture of greatly enlarged well-defined cells that form poorly made out bands. In places these bands show a tendency to interlace. There is little supporting stroma present. Cells show relatively small rounded or elongated nuclei that are moderately hyperchromatic. Cytoplasm is of considerable prominence and is finely granular or finely vacuolated. No mitotic figures are seen. There is no necrosis or evidence of wandering cell infiltration. The portions of tissue are covered in part by intact squamous epithelium although in a few areas the neoplasm has apparently eroded through the epithelium (Fig. 1). At several points superficial epithelial structure descends into underlying tissue in irregular and somewhat disorderly bands. Clumps of epithelial cells are seen in such areas and these clumps are poorly defined by basement membrane. The majority of such deep-lined epithelial cells appear to be stroma cells. Histologically the clumps of cells somewhat suggest malignancy although cytological malignant change is not made out (Fig. 2). Final diagnosis: Myoblastoma in tissue of larynx; superficial epithelial ulceration or erosion with occasional area of beginning epithelial malignant change."

This patient was re-examined two months after the removal of the tumor. It had not recurred, and there was no x-ray evidence of metastases in the neck or lungs.

It will be noted from this report that the tumor consisted of greatly enlarged well-defined cells, some occurring in the form of bands, and that in some areas the tumor had apparently penetrated the epithelial covering (Fig. 2). This produced the appearance of downgrowth of the epithelium so characteristic of epithelioma. This same picture has been described by several pathologists, but according to Ewing,[14] Frenckner[8] and others myoblastomas are not particularly malignant in nature and have but a slight tendency to recur or metastasize after removal.

Careful microscopic study of these tumors is very important, otherwise they may be mistaken for carcinoma with unnecessary radical operation. While these tumors are rarely found in the larynx, the possibility of their occurrence should always be kept in mind.

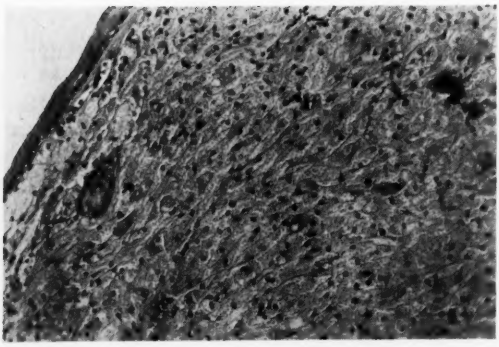

Fig. 1.—Myoblastoma, showing intact superficial epithelium with underlying mass of tumor reaching but not invading the epithelium.

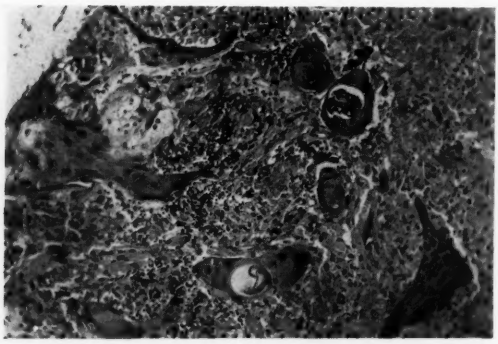

Fig. 2.—Myoblastoma, showing focal epithelial proliferation and possible malignant change in superficial epithelium.

SUMMARY

A brief review of the nature of myoblastoma is presented. Eight cases of myoblastoma of the larynx have been reported in the literature and an additional case is here reported. The importance of the microscopic diagnosis of these tumors is stressed.

707 RACE ST.

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CVI

BRONCHOSCOPY IN THE NEWBORN*

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AND

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Bronchoscopy is seldom indicated in the treatment of diseases of the newborn. However, on rare occasions it is a life-saving procedure in certain cases of atelectasis due to obstruction of the bronchi by body secretions; and at other times it may establish a diagnosis when the pulmonary and radiological signs are confusing.

The procedure should not be undertaken lightly, nor too hastily; but, when gentleness and the proper sized bronchoscope are used, no ill effects have been noted. One of our patients was a premature infant, approximately four pounds in weight, who was not only bronchoscoped twice but had an iodized oil injection in the tracheobronchial tree for radiological diagnosis.

In a personal communication from Dr. Maurice G. Buckles, of Louisville, Ky., he summarizes his experience as follows: "I have been impressed with the efficacy of the procedure and have had no deaths immediately following bronchoscopy, even in the presence of severe cyanosis. Atelectasis is an important cause of breathing difficulty in the newborn, and bronchoscopy is definitely indicated, for prognosis depends a great deal on early recognition and aspiration. I have treated 17 such cases, 11 of which were immediately relieved, two of these being premature."

The indications for bronchoscopy in the newborn are not clear cut. The decision must be made in each case only after careful consideration by the pediatrician and the bronchoscopist, after the usual procedures have failed either to clear the lung or to establish a diagnosis. We do feel, however, that it should be used more frequently, since it has been established that it can be done with safety and benefit to the patient.

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The following case reports illustrate the safety and value of the procedure in these obscure but not infrequent cases.

CASE 1.—(No. 157813.) Massive congenital atelectasis, left lung, from bronchial obstruction by body secretions.

One of twin infants, weighing 5.5 pounds, was brought to the hospital six hours after birth because of persistent cyanosis and dyspnea. The infant was placed in an incubator with an attached oxygen unit and the cyanosis cleared, but returned whenever he was removed from the incubator. A roentgenogram of the chest revealed the left lung to be entirely collapsed with the heart and the mediastinum drawn completely to the left side. The ribs were at a right angle to the spine and the interspaces were narrowed (Fig. 1).

There was no improvement after two days, so the 3.5 mm. bronchoscope was introduced. No marked bronchial obstruction was noted, but suction was applied to the left bronchus for thirty seconds. The procedure lasted approximately three minutes.

Symptomatic improvement was prompt, and a roentgenogram two days later showed normal aeration (Fig. 2). The infant made a complete recovery, although moribund at the time of bronchoscopy.

CASE 2.—(No. 164343.) Massive congenital atelectasis, left lung, due to congenital absence of the left main bronchus.

A premature infant weighing 4.1 pounds was born on Jan. 5, 1941. Because of cyanosis and dyspnea, it was placed in an incubator with an attached oxygen unit, which relieved the cyanosis; however, the cyanosis promptly appeared each time the infant was removed from the incubator. No breath sounds could be heard over the left lung. A roentgenogram showed an opaque left lung with the heart and the mediastinum drawn over to the left side. The left ribs were shelving downward, and there were anomalies of the three upper ribs on each side. Bone anomalies were noted in the lower cervical and upper four or five dorsal vertebrae, the centers of ossification being scattered around without any definite arrangement. It was also noted in the physical examination that there was a supernumerary left ear.

A second roentgenogram two days later showed no change. A third roentgenogram five days later again showed no change and,

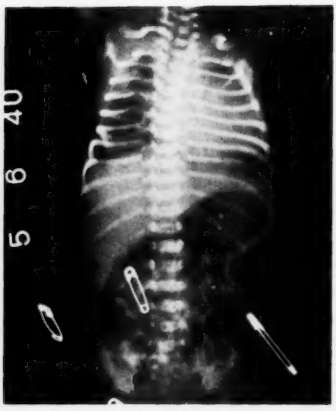

Fig. 1.

Fig. 2.

Fig. 1.—Massive congenital atelectasis, left lung, from body secretions.

Fig. 2.—Same patient as shown in Fig. 1, two days after bronchoscopy. Lung is now well aerated.

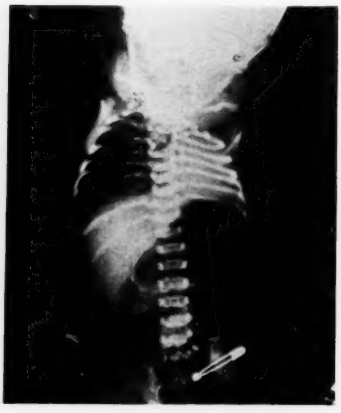

Fig. 3.

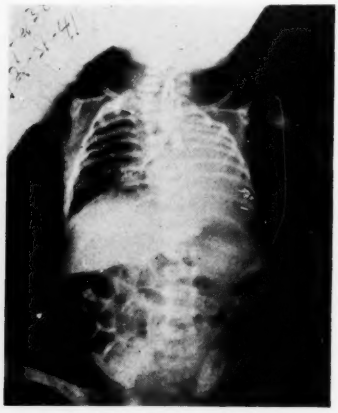

Fig. 4.

Fig. 3.—Massive congenital atelectasis, left lung.

Fig. 4.—Same patient as shown in Fig. 3, with tracheobronchial tree injected with iodized oil, showing absence of left main bronchus.

since cyanosis and dyspnea appeared each time the infant was taken out of the incubator, it was decided to do a bronchoscopy regardless of the anticipated danger, since the baby was premature. It was now felt that there was very little likelihood of the lung becoming aerated without some aid.

On Jan. 14, 1941, nine days after birth, the 3.5 mm. bronchoscope was readily introduced. The larynx was noted to be quite small, much smaller than that of a full-term infant. The trachea was likewise quite small, and the bronchoscope fitted rather tightly. A definite bulge was noted on the left side of the trachea. The trachea was followed downward and only the one lumen was seen, apparently leading into the right lung. No evidence of a left bronchus was visualized; however, due to the small size of the structures, it may have been overlooked. A small amount of mucus was aspirated; no further pathologic changes were noted. After examination the baby was placed in the oxygen incubator apparently no worse for the procedure.

Since the infant's condition was unchanged and no ill effects had been suffered from the first bronchoscopy, it was repeated on Jan. 23, 1941, nine days later. This examination was carried out in a more confident and leisurely manner. Again no evidence of a left main stem bronchus could be found, and it was felt that we were probably dealing with a congenital anomaly. The procedure was well tolerated.

On Jan. 31, 1941, eight days later and twenty-six days after birth, the trachea was injected with 2 cc. of iodized oil through the laryngoscope, and roentgenograms were made. The oil was then removed by suction. The infant stood this procedure well. The roentgenograms showed all of the oil in the trachea, mesial portion of the right lung, esophagus, and stomach. The left lung was still completely atelectatic. At a point where the left main stem bronchus would be expected to start, there was a slight notch. We concluded that there was complete obstruction in the first portion of the left main stem bronchus. Films made the following day showed none of the oil. The lung findings were unchanged.

The infant was discharged nine days later, being then able to stay out of the oxygen incubator, and having gained one-half pound. He has been followed in the out-patient department and, outside of occasional periods of cyanosis and a progressive scoliosis, is doing quite well. The lung condition shows no change. The child is now 16 months old.

Fig. 5.

Fig. 5.—Massive congenital atelectasis, left lung, from absence of left main bronchus in an 11-year-old child.

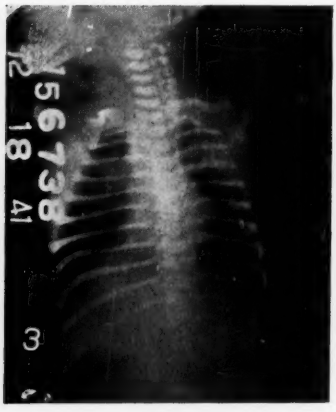

Fig. 6.

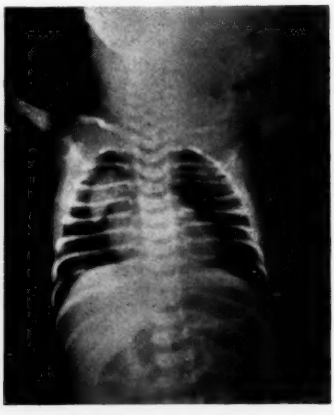

Fig. 7.

Fig. 6.—Partial congenital atelectasis, bilateral, with pneumothorax.

Fig. 7.—Partial congenital atelectasis, right upper lobe, and pneumothorax.

CASE 3.—(No. 167887.) Massive congenital atelectasis, left lung, due to congenital absence of left main bronchus.

This patient, an 11-year-old boy with the same anomaly, is reported with the preceding cases to show what may be expected in later years and because of its rarity and interest.

On April 28, 1941, an 11-year-old boy was admitted for study because of unexplained atelectasis of the left lung and scoliosis. This boy had been recently treated for measles, from which he had made an uneventful recovery. However, during this attack, the marked scoliosis and atelectasis were noted. This patient had had the usual childhood diseases, but had otherwise been in good health. He had been observed by other physicians, and a tonsillectomy and adenoidectomy and circumcision had been performed at the age of 8 years. No mention of the condition in the left lung had ever been made, although the pigeon breast had frequently been referred to and there was a history of respiratory difficulty after birth.

Physical examination was entirely negative except for the chest condition. Roentgenograms on April 28, 1941, showed complete atelectasis of the left lung. A part of the right lung was seen on the left, and the heart and the mediastinum were markedly drawn to the left side. The right lung was emphysematous. The ribs on the left were drawn closer together, producing a right lateral scoliosis. The left diaphragm was elevated. No radiopaque foreign body was present. No cause for this atelectasis was apparent.

A diagnostic bronchoscopy was done on May 2, 1941. The trachea was so small that a 5 mm. bronchoscope was passed with some difficulty; in other words, the trachea was approximately the size of the right main stem bronchus. The bronchoscope passed directly into the right main bronchus, and its main divisions were identified. There was no evidence of the lumen of the left bronchus. At the approximate level of this bronchus, there was a ridge across the left wall which possibly represented the carina. It is believed that we were dealing with a congenital absence of the left main stem bronchus. Iodized oil was injected, but the injection was unsatisfactory, and no additional information could be gathered from the roentgenogram.

The family would not permit further study, so the patient was discharged the following day. He has remained well since, and has been fitted with proper orthopedic appliances.

CASE 4.—(No. 175024.) Partial congenital atelectasis and pneumothorax.

This infant was born on Dec. 17, 1941, and since cyanosis was marked, was placed in an oxygen unit, but with only slight benefit. Roentgenograms on Dec. 18, 1941, showed no apparent obstruction in the pharynx or trachea. There was marked emphysema of both lower lobes and a small pneumothorax pocket in the lateral portion of the left costophrenic angle, probably due to a rupture of an emphysematous bleb. In contrast, the upper lobes were increased in density with a string-like accentuation of the bronchial markings, suggesting a partial atelectasis. The heart appeared normal.

Since the infant's condition was critical, a bronchoscopy was requested, in hopes that aeration could be obtained. On the same day, the 3.5 mm. bronchoscope was introduced and both main stem bronchi were aspirated. A small amount of secretion was present, but no bronchial obstruction was noted. Two days later there seemed to be slight improvement, so the procedure was repeated and mucus was aspirated from both main bronchi. Breathing seemed to be easier afterward; however, death followed several hours later, but was in no way associated with bronchoscopy. Unfortunately, no autopsy was obtained.

CASE 5.—(No. 177720.) Partial congenital atelectasis with pneumothorax.

This infant was brought into the hospital five hours after birth, on March 22, 1942, because of persistent cyanosis. A roentgenogram revealed atelectasis in the right upper lobe and partial pneumothorax. He did not improve under oxygen, and his course was progressively downhill. A bronchoscopy was done the day after admission, but no obstruction to the airway was noted, and no secretions were present. The whole examination lasted about one minute; the infant was returned to its oxygen incubator, but died several hours later. Unfortunately, no autopsy was obtained.

Bronchoscopy was of no value in these last two cases, because the congenital condition was not one of atelectasis due to body secretions. However, the procedure was well tolerated and, since we were faced with a hopeless condition, bronchoscopy was advised and done with the hope that better aeration might be obtained and the infants saved.

CONCLUSIONS

Bronchoscopy can be carried out in newborn infants with safety, and at times is a life-saving procedure. At other times it aids in establishing a diagnosis. If employed more widely for all types of congenital atelectasis, the indications and contra-indications will be sooner understood and many lives will be saved.

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CVII

FURTHER OBSERVATIONS ON TUBERCULOUS ENDOBRONCHITIS

KENNETH A. PHELPS, M.D.

MINNEAPOLIS

The present lively interest in bronchial tuberculosis is indicated by the many recent publications on the subject by pathologists, roentgenologists, chest physicians, surgeons, and bronchologists. Most of these authors agree on the symptomatology and diagnosis, but many hold directly opposite views regarding the treatment.

The bronchoscopist who is accustomed to treating patients whose chief difficulty is their bronchial condition will soon realize that this is not true when he deals with bronchial tuberculosis. In patients with bronchial tuberculosis the major problem is the general disease. The bronchial lesion is of importance because it alters the prognosis and complicates the treatment of the pulmonary tuberculosis. The final outcome will depend more upon the tuberculosis of the lung than upon that of the bronchus.

Pathologists agree that in most cases of cavitation some degree of endobronchial ulceration is present in the draining bronchus. The infection spreads by direct extension along the endobronchial wall as a submucous lesion. This is rarely seen bronchoscopically, as most of these lesions heal spontaneously without producing symptoms. The factors that determine whether healing or progression is to occur are the virulence of the organism and the resistance of the host.

When the submucous lesion breaks down into an ulcer and granulation tissue forms, the bronchus becomes occluded and the well-known symptoms of obstruction develop. This is the stage most commonly seen at bronchoscopy.

Some, but not all, ulcerogranulomatous lesions heal by scar tissue formation and a cicatricial stenosis of the bronchus results. This stage is also frequently encountered by the bronchologist.

Dr. George Higgins, the pathologist at Glen Lake Sanatorium, has found superficial ulcers, almost miliary in type, in the trachea and bronchi in 20 patients at post mortem examination. These were

in hopelessly advanced cases, mostly men, who had had no symptoms of bronchial obstruction and no bronchiectasis. This is a terminal lesion and is not seen at bronchoscopy.

In our series of 67 cases of endobronchial tuberculosis, 65 were women and only 2 were men. There is no reasonable explanation of this fact.

Tuberculosis is rarely primary in the bronchus. The diagnosis of pulmonary tuberculosis has been made and treatment has usually been instituted before endobronchial tuberculosis is discovered. However, some cases might be confused with endobronchial tuberculosis in which the bronchologist makes the diagnosis by removing tuberculous tissue from a bronchus. This tissue is generally a gland which has ruptured into the bronchus. As a rule no permanent injury to the bronchus results. When the gland recedes, the bronchus generally becomes normal. Rarely the cartilaginous rings may become absorbed and the weakened bronchial wall may collapse. A gland rupturing into a bronchus should not be considered true endobronchial tuberculosis.

The clinical diagnosis is based on the persistence of symptoms and signs in spite of adequate control of the pulmonary lesion. It is this circumstance which makes one realize that symptoms believed to be due to parenchymal disease may be due to bronchial lesions instead.

Persistent positive sputum may result from an ulcer in the bronchus, as we have seen such cases become negative after treating the ulcer locally. Sputum retention with fever, cyanosis, and cough is due to temporary total occlusion of the bronchus. The symptoms recede when drainage occurs, either spontaneously or following bronchoscopy.

Wheezing and dyspnea, unexplained by the pulmonary lesion, also suggest bronchial obstruction.

The x-ray findings are those of bronchial obstruction: emphysema, atelectasis, "tennis ball cavities" with a fluid level and the size varying from day to day. Bronchograms and laminograms may demonstrate a narrowed bronchial lumen.

In our experience the bronchoscopic diagnosis corroborates the clinical and x-ray diagnoses in about 75 per cent of the cases. When the clinical and x-ray evidence is convincing, a negative bronchoscopy merely locates the disease beyond the field of the bronchoscope.

We do not do routine bronchoscopic examinations. Our patients are carefully selected and have suspected bronchial lesions.

Bronchiectasis was proven by bronchogram or postmortem examination to be present in 80 per cent of our cases. Sixteen per cent more had definite symptoms, though without proof. Bronchiectasis is the end result of chronic infection of the bronchial wall with destruction of its elastic tissue plus bronchial obstruction with emphysema, or with atelectasis, and traction on the bronchial wall from altered intrathoracic pressure. In my first paper on this subject, presented before this society in 1936, it was stated that a nontuberculous bronchiectasis might be caused by a tuberculous stenosis of the bronchus. Since then it has been observed that pathological evidence is in favor of the bronchiectasis being tuberculous as well as the stenosis.

Bronchoscopy is not recommended as a routine procedure in all tuberculous patients. It is advisable that patients have a bronchial examination before any irreversible collapse therapy is instituted. The bronchologist is impressed with the remarkable control over their throats which sanatorium patients have. They are trained to control their cough and this control helps the bronchologist immeasurably. Simple equipment is all that is required and in most cases no laryngoscope is used to expose the larynx. We have found that the mask used by industrial workers to protect the eyes and face is very satisfactory for the bronchoscopist.

Ulcerostenotic lesions do not behave in a uniform manner. Some heal quite promptly with treatment, while others heal without treatment. Some do not heal with or without treatment and the ulcerogranuloma may continue for years. This explains the variation in opinion regarding the results of treatment. We are unable to predict with any certainty what the course of any individual patient will be, so we treat them all until we find out.

It seems to us that local treatment of the ulcerostenosis with 30 per cent silver nitrate helps many patients. Some become sputum-free, at least for a while; some breath better, have less frequent retention attacks and apparently heal quicker.

When the granulation tissue can be removed by forceps, it is done even though it is realized that a complete removal is impossible. Improvement in the airway sometimes results in better drainage.

Local treatment of the fibrotic stenosis by dilatation is not satisfactory in our experience. In fact, dilatation may result in a

temporary complete occlusion of the bronchus with very unpleasant results.

Before any collapse measure is undertaken, the condition of the bronchus should be recognized. Bronchoscopy is indicated when there is a suspicion of a bronchial lesion. It should be realized that, if obstructive bronchitis is present, it will result in some form of permanent damage to the bronchus, accompanied by a permanent lung change, bronchiectasis.

Permanent therapy in some form would seem logical. Control of the parenchymal disease may not affect the endobronchial tuberculosis, or the bronchitis may become healed even though the pulmonary lesion persists. This explains why authors are not agreed on the results of collapse therapy. Some patients do well on long-continued pneumothorax; others do well on thoracoplasty; while others need a lobectomy or pneumonectomy. The chest surgeon should make the decision as to which type of collapse therapy is indicated.

In our series of cases in five years, 31 per cent are dead, 63 per cent have chronic disease, and 6 per cent are well.

If the bronchiectasis is of moderate degree, the patient may remain in a reasonably good state of health for an indefinite period but, when the bronchiectasis is advanced, the patient is permanently incapacitated.

CONCLUSION

1. The bronchologist's viewpoint must be to "treat the patient rather than the bronchus."

2. A gland ruptured into a bronchus should not be considered a variety of endobronchial tuberculosis.

3. The pathologist sees terminal lesions which the bronchologist does not see.

4. Bronchiectasis is proven to be present in 80 per cent of the cases, and probably is present in 96 per cent.

5. Local treatment may help and should be tried.

6. Collapse therapy should be selected for the individual patient. Some do well on pneumothorax, some on thoracoplasty, and others require lobectomy or pneumonectomy.

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CVIII

BRONCHIAL INVOLVEMENT IN PURPURA HEMORRHAGICA

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Purpura hemorrhagica with pronounced symptoms of bronchial involvement is uncommon. We frequently see skin manifestations of this disease, but severe hemoptysis without other demonstrable changes is very rare. Therefore, this case is being reported to call attention to the possibility of profuse pulmonary hemorrhage resulting from such a disease.

REPORT OF A CASE

O. A., a white man, 31 years of age, married, and a salesman, was referred to me on Dec. 30, 1940, for consultation in regard to frequent and severe attacks of hemoptysis. He was sent to the hospital for a thorough physical examination and bronchoscopy.

The patient gave a history of coughing up about a cupful of bright, red blood in June, 1940, following a strenuous game of baseball. At first the blood flow was rather free, but later the blood became clotted and was mixed with mucus. Following this attack, he continued to expectorate small clots of blood for several days. During this time he had no other noteworthy symptoms. After the onset, the patient had six or seven attacks of hemoptysis, each lasting for three or four days. The last attack occurred about one month before admission to the hospital. Bleeding at this time occurred after strenuous exercise and continued for several days, being aggravated by the slightest cough.

The patient also gave a history of having received a severe injury to his back in 1928, for which he was confined to bed for several months. He complained of some pain in his back at various times, but no serious trouble developed until 1934, when he began passing large quantities of bright red blood in his urine. At that time he was placed in the hospital and given a thorough examination, including a pyelogram, and was told that he had only one active kidney, the other one having become atrophic as the result of his accident.

After discharge from the hospital at this time, he had no particular trouble except for occasional attacks of pyuria until the start of the hemoptysis in 1940.

Before seeing me he had consulted several prominent internists, but a definite diagnosis had not been made and he continued to cough up blood-stained mucus from time to time. Tuberculosis was suspected, but on all occasions examination of the sputum by smear and inoculation of media failed to reveal any acid-fast bacilli.

While in the hospital, the patient was given a thorough physical examination. His chest was well developed, and resonant throughout; expansion was good; there was no lag; the bases descended well. The breath sounds were vesicular, and there were no râles. The heart was normal; the blood pressure, 124/30.

Roentgen examinations of the chest and abdomen were reported as follows:

"The chest shows considerable increase in size and density of the hilar regions, with exaggeration of the bronchovascular markings extending into the right base. A small amount of stringy infiltration appears to be present about the descending trunks on the right; however, this is not definite from this examination. The lungs are otherwise clear. The diaphragm, heart, and vessels are normal. The abdomen shows both kidneys poorly demonstrated. No radiable urinary calculi are visualized. A rounded area of increased density measuring about 6 mm. in diameter is present in the left side of the pelvis. This has the appearance of a phlebolith."

Impression: Possible infiltration about the descending trunks on the right with increase in the hila regions.

"The kidney, pelveo ureters and bladder following intravenous injection of diodrast are fairly well demonstrated. The calices appear slightly blunted. The right ureter is slightly dilated in its middle third. The renal pelvis is not abnormal. The right kidney is well visualized on this examination and appears normal in contour and position, but is slightly enlarged. The left kidney is not visualized and no opaque medium is present on this side. Impression: Slightly enlarged right kidney with early hydronephrosis; however, the changes may be compensatory in nature. There is a possible hydroureter, middle third. The left urinary tract was not visualized. A retrograde examination is advised.

"An x-ray of the paranasal sinuses shows a slight clouding of the maxillary antra. The remaining sinuses are normally developed and aerated. The calvarium is not unusual. The pineal gland appears normal. The pituitary is not calcified and is situated in its normal position."

Impression: Possibly slight maxillary sinusitis.

Laboratory findings: NPN, 35; sugar, 127; uric acid, 4.3; chlorides, 280; CO_2, lost.

Agglutination Titer—serum dilutions with antigen.

	1-20	1-40	1-80	1-160	1-320
Eberthella typhosa	0	0	0		
Salmonella paratyphi	0	0	0		
Salmonella schottmuelleri	0	0	0		
Brucella abortus, suis, melitensis	0	0	0		
Pasteurella tularensis	0	0	0	0	0
Rickettsia (Weil-Felix Reaction)	0	0	0	0	0

Blood count: Hb., 84%, Sahli, 12.2 grams; RBC 4,420,000; WBC, 6,200; PMN, 57%; filamentous, 56; nonfilamentous, 1; lymphocytes, 39; monocytes, 1; eosinophils, 2; basophils, 1; platelets, 712,800; reticulocytes, 4.2%; volume index, .99; M. c. v., 86.4 cubic microns; prothrombin time, 19 seconds; color index, .95.

Sedimentation test: First reading, 28 mm.; corrected reading, 15 mm.; blood volume, 38 mm.

Wassermann (Kline): Negative.

Sputum: No acid-fast bacilli observed. Media inoculated.

No definite pathological lesions were found, except the kidney changes recorded and the bronchoscopic findings which are as follows:

The bronchoscopy was performed under local anesthesia. The mucosa of the mouth, pharynx and larynx were of normal color; no hemorrhagic spots were found; no edema was noted. There were a

considerable amount of blood-stained mucus and old clots on the tracheal surface. Both of the lower bronchi contained a rather large amount of blood-stained mucoid exudate with some clotted blood. This was aspirated but no free bleeding surface was found at this examination. Lipiodol was instilled as bronchiectasis was suspected. No evidence of a new growth could be made out in any of the bronchi. Material was taken for cultures and smears, but no acid-fast bacilli were found.

An x-ray report, following the instillation of lipiodol, revealed that the bronchi were nicely visualized and appeared entirely normal throughout. There was no evidence of bronchiectasis.

The patient was allowed to go home and about fifteen days later developed another attack of gross hematuria. At this time he had a chill and fever which lasted a few hours. He was returned to the hospital again where he was given Clottagen and vitamin K. He continued to have some hemoptysis and another bronchogram was made after instillation of lipiodol, but no bronchiectasis was found.

After leaving the hospital, the patient returned to his home near Washington. Nothing was heard from him for several months, but in November, 1941, a friend called me up and stated that Mr. A. had had another severe attack of bleeding. He had been sent to a hospital in Washington, where he was "dying with cancer of the lung."

Shortly afterward death resulted; however, an autopsy did not show any malignant changes in the lungs nor any tuberculosis, but it did show purpura hemorrhagica.

Purpura hemorrhagica has been defined by Wintrobe[1] as follows: "This is a condition of unknown etiology which is characterized clinically by petechiae or ecchymoses in the skin as well as by hemorrhage from mucous membranes and into various tissues. The onset and course of the disease are variable and may be marked by spontaneous remissions and relapses. The skin lesions are not associated with erythema, swelling or inflammation. The platelet count is usually reduced, the bleeding time is prolonged, the clot nonretractile and the tourniquet test positive, whereas the coagulation time is essentially normal. There is no anemia except that which can be attributed to blood loss. Leukocytic changes are insignificant."

While epistaxis is a frequent symptom and bleeding from the mucosa of the mouth and gums often occurs, a review of the litera-

ture does not show hemoptysis as a characteristic symptom of the disease.

This case has been reported on account of the unusual bronchial involvement, a condition which should be considered in all undiagnosed cases of severe pulmonary hemorrhage.

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CIX

HEMORRHAGE OF THE RESPIRATORY TRACT: A BRONCHOSCOPIC PROBLEM

ROBERT E. PARRISH, M.D.

SAN ANTONIO

Hemorrhage from the respiratory tract is a very common symptom caused by many different conditions. These conditions can be divided into two main groups. First, lesions arising in the respiratory tract or lung structure, and, second, lesions arising outside the respiratory tract which, by growth or other means, invade the respiratory tract. A third condition which might also be mentioned is trauma, direct or indirect, involving the respiratory tract or lung structures. The importance of hemorrhage from the respiratory tract, both to ourselves and to our patients, is so great that every effort should be made to determine its cause and location so that proper treatment can be instituted. In some cases the cause and location of the bleeding can be easily determined, while in others the difficulty is so great that it can be considered one of the greatest problems with which the bronchoscopist is confronted. A large number of patients coming to us for bronchoscopic examination present this symptom.

In many cases of respiratory tract hemorrhage the cause or probable location can be determined by the chest specialist or internist. Often we are called upon to verify the diagnosis, especially in cases with doubtful clinical pictures. In some instances bronchoscopy is the only possible means of making the diagnosis. Fortunately, the diagnosis can be made in a fairly large percentage of cases. However, in some cases, even after all means of examination have been exhausted, the diagnosis still remains uncertain.

In 1936, before this Society, Arbuckle[1] and Myerson[2] presented articles on this subject to which I wish to call attention. Arbuckle listed the cause of respiratory tract hemorrhage in their order of frequency, as follows:

(1) Tuberculosis. (2) Ulcer associated with bronchiectasis or abscess. (3) Tumors. (4) Aneurysm. (5) Broncholith. (6) Unknown causes. A careful consideration of these causes should be

borne in mind when any case is being considered. It might be well to use the term "other causes" rather than "unknown causes," because as time goes on fewer cases in which the cause is unknown are encountered.

When a patient is referred for bronchoscopy, all available aids to diagnosis should be employed before bronchoscopy is done, providing the case permits such delay. In the first place we should examine the patient carefully to determine that the blood is coming from the respiratory tract. Examination by x-ray and chest specialists should be carried out. The usual laboratory examinations should be made.

The amount of bleeding from the respiratory tract is of little significance in diagnosis, as a severe lesion or pathological process may cause little or no bleeding, while an insignificant or benign lesion may cause quite frequent or extensive hemorrhage. The character of the bleeding gives some indication of the kind of pathology present. Free bleeding, of small or large amount not mixed with purulent discharge, does not indicate the presence of much infection, but the cause may or may not be associated with some other definite lesion. If the blood is fairly well mixed with mucopurulent discharge, it is probably coming from an acute infectious process in the lung, from bronchiectasis, or from a lung abscess. Blood mixed with pus in large amounts is more often associated with lung abscess or bronchiectasis. In each case there is usually a fairly characteristic odor which we are all familiar with and which may be an aid in the diagnosis of the pathology present. If there is pus and blood in large amounts without a distinctive odor, the likelihood of its coming from the pleural cavity or subdiaphragmatic space should be considered.

Bronchoscopic examination should not be done for diagnosis during an acute respiratory infection. It is best also to wait a week or ten days following extensive hemorrhage before the bronchoscope is passed. However, a small hemorrhage is not a contraindication to bronchoscopy. As a matter of fact, a small amount of bleeding at the time of the examination may be a distinct aid in the location of the bleeding point. If the hemorrhage is too great at the time of bronchoscopy it obscures the view and also interferes with the obtaining of a biopsy if a diseased process is found. Severe hemorrhage is difficult to control even if we know where it is coming from. Arbuckle used a string tampon in some of his cases but he advised against leaving the string tampon in place more than twenty-four hours on account of the development of pneumonia. Hemorrhage from the

lung may be so severe as to cause death even in patients in whom little or no pathology can be found. Therefore, no case should be lightly regarded. In some cases the lesion causing the hemorrhage may also be fatal but with proper diagnosis and proper treatment we undoubtedly could save some of the patients that are now lost. Severe hemorrhage coming from the lung structure outside the trachea or main bronchi can best be controlled by pneumothorax. The lung should be collapsed on the side from which the blood is coming. It is necessary to be certain which side the hemorrhage is coming from so that the chest specialist will be able to collapse the lung on the affected side.

If it is possible to make a correct diagnosis and if the patient is suitable for treatment by bronchoscopy, the responsibility for such treatment rests with us. But any diagnosis or recommendation must be correct so as not to mislead the chest specialist or chest surgeon in the management of the case.

Other physicians, as well as our patients, should be informed that a bronchoscopy does not always solve the problem. It is not always possible to make a diagnosis or locate a lesion causing the bleeding. Bronchoscopic examination consists only of a direct inspection of the larynx, trachea and larger bronchi. If no pathology is found and no specimen is obtained, the lesion may be farther out in the lung tissue, and the examination thus is of a negative rather than a positive value. Repeated bronchoscopic examinations are sometimes necessary and some patients must be kept under observation for a long period of time before a positive diagnosis is possible. If a definite lesion is found, a satisfactory biopsy may be all that is necessary. If a lesion is not found, collection of a specimen of the bloody discharge, if present, and bacteriological and pathological study may give the answer. A bloody discharge can be examined after it is centrifuged and embedded in paraffin for section and pathological study with very much the same result as a biopsy. Direct inspection of the larynx, trachea and bronchi is often sufficient for a fairly definite diagnosis if the lesion is present in the area inspected. Certainly we can tell ulcers from tumors and inflammatory processes from non-inflammatory. However, to verify the findings, biopsy is of enormous value. It is much easier to diagnose the cause of a hemorrhage in patients with physical or x-ray findings. The reason for this is the fact that these findings reveal the location of the diseased process and often give a fair idea of what it might be. This makes the problem much easier than when it is necessary to

explore the entire respiratory tract searching for a process so small as to produce no x-ray or physical findings.

Three cases are reported. The first one is reported as an unusual case of respiratory tract hemorrhage, the second is a case as yet unsolved but perhaps is secondary to trauma of the chest, and the third is a case in which trauma was the probable cause but in whom it was impossible to determine the site of bleeding even though the patient died and an autopsy was done.

REPORT OF A CASE

R. V., white, male, aged 34, was admitted to hospital on Sept. 19, 1941. The chief complaint was a gradual loss of weight for the past six months, cough and expectoration of blood daily for the past three months. There was pain in the right side of the chest. His normal weight was 132 pounds; his present weight, 102 pounds. Admission diagnosis was possible cancer of the right lung.

A physical examination showed the right lower chest to be dull and the respiratory movements restricted. He expectorated blood-tinged sputum. The examination was otherwise negative. The patient was running an irregular septic type temperature. The urine examination was normal. The red blood count was 1,900,000; the hemoglobin, 4.5 gms. The blood Wassermann was negative. The sputum examination was negative for tuberculosis.

An x-ray examination of the chest showed: thickened pleura and elevation of the diaphragm to level of fourth rib anteriorly, no apparent enlargement of the mediastinum, and an old chronic healed tuberculosis of the right apex.

A bronchoscopic examination revealed no pathology in the trachea or the bronchi. A purulent discharge was draining into the right main bronchus through the secondary bronchi near the midline in the direction of the dome of the diaphragm. At the time the bronchoscope was introduced the patient coughed and a large amount of creamy, chocolate-colored discharge began to flow from the trachea. The quantity was so great, a constant suction was necessary for perhaps five minutes, during which time an estimated one-half pint or more of discharge was aspirated. There was no characteristic odor to this discharge. A bronchoscopic diagnosis of a ruptured abscess from the pleural cavity or subdiaphragmatic space, possibly a liver abscess, was made. The discharge was examined without a positive diagnosis as to the etiology of the abscess. Other laboratory examinations were also negative. The patient was placed on emetine

hydrochloride injections for a ten-day period and given a blood transfusion. He began to improve rapidly and was discharged from the hospital Dec. 1, 1941. A re-check of his condition on May 1, 1942, found him with normal weight and free of symptoms. A lipiodol injection indicated a normal bronchial tree. The pleura was thickened over the lower lobe and the diaphragm was elevated, probably from adhesions.

This case is reported as an unusual cause of respiratory tract hemorrhage. The final diagnosis was subdiaphragmatic abscess which had ruptured into the pleural space and subsequently drained through the bronchus from the right lower lobe.

REPORT OF A CASE

R. F., white, male, aged 40, weight 200 pounds, had a negative family history. His past history showed he had the ordinary diseases of childhood. He had had an appendectomy and a tonsillectomy four to twelve years ago. Two of his ribs were broken two years ago. The patient states that he has felt very tired for about one month. About two weeks ago he began to expectorate blood. This occurred about one week after falling from a horse and slightly injuring his chest. He had pain occasionally in the chest and a warm sensation at times. Expectoration of blood for about a month occurred freely during the day and night. His temperature was normal. The sputum examination was negative for tuberculosis. An examination of his nose and throat was negative. The blood could be seen coming from beneath the vocal cords with the laryngeal mirror. An x-ray examination of his chest failed to reveal any pathology. A physical examination of his chest was normal. Bronchoscopy was done on August 18, 1941. A small amount of thick semiclotted blood was seen in the trachea and both main bronchi. No bleeding point, no tumor, no inflammation, or no ulceration was found to account for bleeding. The location of the hemorrhage was not determined.

This patient continued to expectorate blood. He had a bronchoscopy in another city about one month later. A report from the doctor doing the bronchoscopy indicated he found no lesion, but he stated the blood was coming from the left lower lobe. A diagnosis as to the cause was not made. The patient continued to expectorate blood and had two bronchoscopic examinations in a third city; there the diagnosis was that the blood was coming from the right lower lobe and was probably associated with bronchiectasis and it was recommended that, if he got worse, the right lower lobe

should be removed. The patient has continued to expectorate blood since that time, about nine months, but the amount and frequency is gradually decreasing. He refuses to have further bronchoscopic examinations and thus far the diagnosis as to the cause of the bleeding is undetermined. There is a question as to location of the hemorrhage as one bronchoscopist places it on the left side and one places it on the right; whereas, in my examination, I frankly confess, I was unable to determine with any degree of certainty from which side the blood was coming.

Because of the uncertainty of the diagnosis by bronchoscopic means and because of the absence of x-ray and physical findings, the chest specialist managing this case is greatly handicapped in formulating any definite lines of treatment. The diagnosis was respiratory tract hemorrhage, location and cause uncertain.

REPORT OF A CASE

B. B., white, male, aged 16, was admitted to the hospital Nov. 29, 1941. One week before admission to the hospital the patient strained himself while trying to lift up the bumper of one automobile to free it from another. Immediately he began to cough and had a large pulmonary hemorrhage. Some bleeding continued off and on for one week until he was admitted to the hospital by ambulance. The patient had had no previous serious illnesses and no accidents.

The physical examination showed that his blood pressure was 140/68, that he was well developed and well nourished. The upper respiratory tract was normal. "The lung expansion is free and equal. Increased breath sounds over area medial to and below the nipple on the right. There are no definite râles at present." The abdomen was negative; the urine examination, normal. Red blood count was 3,680,000; white blood count, 11,200; coagulation time, 4 minutes. Blood Wassermann was negative. Repeated sputum examinations were negative for tubercle bacilli. An x-ray examination of chest showed the lung fields clear. The patient had a temperature up to 101° for two days after admission. The temperature then remained normal.

Five days after admission bronchoscopy was done with the following findings: "There is no evidence of new growth in the trachea or the main bronchi. There is no evidence of purulent or mucopurulent discharge. No definite site where a large hemorrhage might have originated is seen. There is a mild granular condition of the membranous lining of the right main bronchus which bleeds a small

amount on contact with the bronchoscope. This condition indicates a subacute or chronic inflammatory condition. This is not believed to be the cause of the severe hemorrhage."

The patient was discharged from the hospital and he returned to his home in a small town about 150 miles away. About one week after he reached home, the patient again had a fairly large hemorrhage. Bleeding stopped for two days at which time, while he was sitting up in bed eating his supper, he suddenly began to hemorrhage severely and died within 40 minutes. The patient was being treated in a small, community hospital at the time. The doctor in charge gave him one transfusion, artificial respiration, oxygen, etc., but to no avail. An autopsy was obtained and his doctor reported as follows:

"The gross findings showed large areas of ecchymosis on the external surface of the lungs, which were apparently secondary to the hemorrhages into the bronchi. I could not find any gross pathology and could not find the vessel from which he was hemorrhaging."

Tissue was removed for pathological study and the report was as follows: "Sections of the lung tissue show some free blood in the alveoli. This is apparently recent as there is very little cellular reaction attending the presence of the blood. Some of the sections from a larger bronchus show an acute inflammatory reaction in the bronchial mucosa with necrosis and erosion of the bronchial mucosa. Sections do not include any definite blood vessel erosion. The submucosa in these bronchi is infiltrated mostly with lymphocytes and a few polymorphonuclears. Sections of the lymph nodes show some hyperplasia, especially of the lymphocytic element. We see no evidence of any tumor or any tuberculous process."

DIAGNOSIS: "There is hemorrhage into the lungs; non-specific bronchitis with erosion of the mucosa; and nonspecific hyperplastic lymphadenitis."

CONCLUSION

1. Respiratory tract hemorrhage is caused by many different conditions in or about the respiratory tract.

2. Bronchoscopy is a definite aid in the diagnosis of the location and cause of this hemorrhage in many cases.

3. All present methods of examination may fail to reveal the location or cause of the hemorrhage in some cases.

4. Fatal bleeding may occur without sufficient definite pathology to determine the locations of the bleeding point.

I am indebted to Dr. R. G. McCorkle of San Antonio, Texas, for the reference of cases Nos. 2 and 3 and for portions of the histories of these cases.

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CX

FOREIGN BODY IN THE UPPER LOBE BRONCHUS

ROBERT L. MOORHEAD, M.D.

BROOKLYN

Foreign bodies in the upper lobe bronchi fortunately are rare but they have always constituted a major problem in bronchoscopy. The use of the curved upper lobe forceps in combination with the biplane fluoroscope has solved the mechanical problem in most cases. However, the biplane fluoroscope may not always be available and, although this greatly increases the difficulty, it may not be an insurmountable drawback. It has long been observed that in cases of pulmonary collapse the upper lobe bronchus is not only displaced downward but is rotated, so that the angle which it forms with the main stem bronchus is greatly reduced. The knowledge that this displacement occurs greatly aided in the solution of the following case.

REPORT OF A CASE

In November, 1941, a young draftee, 21 years of age, was referred by his draft board to the Long Island College Hospital because of a chronic cough. In the course of his examination an x-ray was taken of his chest which revealed a nail in the right upper lobe bronchus. This was identified as a lather's nail. As the patient had not worked as a lather for over two years the nail had been in the bronchus for at least this long. As can be seen by the x-ray, there was remarkably little pulmonary irritation considering the length of time, but this was apparently due to the fact that a lather's nail has a special coating which prevents erosion and was thus probably much less irritating than an ordinary wire nail. He was admitted to the hospital on November 27, and on December 1 a 9 mm. bronchoscope was passed under local anesthesia, but nothing was accomplished. He was again bronchoscoped on December 4 and 15 and on January 19, each time with the assistance of a single plane fluoroscope. Of course the ideal method would have been to have used a biplane fluoroscope, but such a machine was not available; so only the single plane fluoroscope was used. During the several bronchoscopies, the nail was grasped on three separate occasions but the long axis of the forceps did not coincide with that of the nail.

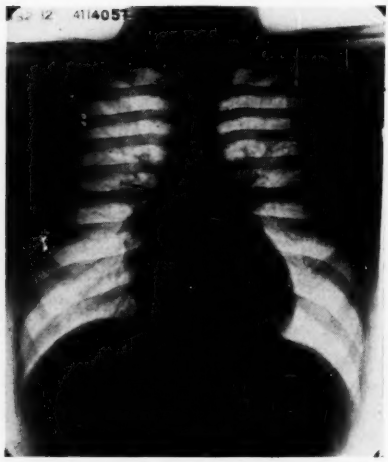

Fig. 1.—Roentgenogram showing nail in right upper lobe bronchus.

Fig. 2.—Roentgenogram showing nail in the right upper lobe bronchus; after artificial pneumothorax.

The resulting angle caused the point of the nail to impinge on the bronchial wall on any attempt at manipulation. The change in position of the upper lobe bronchus in cases of pulmonary collapse having been frequently noted, it seemed that this change might be of aid in solving this problem. Accordingly, on February 3 Dr. Richard Bennett of the Medical Service introduced 450 cc. of air into the right pleural cavity, and an x-ray of the chest now showed the nail to be about an inch lower and at a decidedly more favorable angle. On February 5 a bronchoscope was again introduced with the aid by the single plane fluoroscope, and the nail was grasped by a Tucker forceps. After rotation of the forceps, the nail was easily withdrawn into the lumen of the tube and was removed. At no time during this procedure was the nail visualized in the bronchus. There was no reaction from the bronchoscopy and the patient was discharged from the hospital on February 10 and arrangements were made for a follow-up by the out-patient service.

125 Remsen St.

CXI

A BRONCHOSCOPIC DIAGNOSTIC SIGN FOR LESIONS OF THE PLEURAL CAVITY

M. C. Myerson, M.D.

New York

Several years ago I chanced upon a peculiar bronchoscopic picture. A man, 37 years of age, was referred for bronchoscopy because he had suffered with cough and dyspnea for 15 years and was having hemoptyses, which had occurred during the past four years. A diagnosis of tuberculosis had been made. He did not have any fever and had not lost any weight. His principal complaint was that he felt a sensation of constraint in both sides of the lower part of his chest. His sputum was streaked with blood, except in the early morning when he would cough up a thick dark plug.

On examination, there was flatness on percussion and total absence of breath sounds over both lower lobes. A Wassermann test of his blood was negative. X-ray studies were inconclusive. Bronchoscopy was requested in order to explain the lack of aeration of the lower lobes of the lung and the hemoptysis.

Bronchoscopy revealed a jelly-like tumefaction which completely obstructed the introitus of the left lower lobe bronchus. An application of adrenalin chloride solution caused the immediate disappearance of the swelling in the primary bronchus, but a similar tumefaction could be seen in the branches beyond. The tumefaction, which probably extended into the periphery, interfered with aeration of the corresponding lung.

On the right side there was a similar involvement of the posterior branch of the lower lobe bronchus. Here the obstruction was complete.

Both pleural cavities were aspirated and fluid obtained from each. Bacteriologic study disclosed that the fluid was sterile. When the needle was introduced into the left side of the chest, it encountered a stiff resistant wall and a definite scratching sound was heard. A diagnosis of calcification of the pleura was made.

The patient related that some years before he had developed a serious disease of his lungs while aboard a freighter. His illness was

characterized by fever, cough, and pain in the chest. Probably he had had pneumonia, which was followed by empyema. Since he had had no medical attention at the time, it would appear that he had a bilateral empyema, following which there occurred a calcification of the pleurae and a retention of exudate which eventually became sterile.

This experience demonstrated to me for the first time that intense, passive congestion of the bronchial mucosa could occur as a result of a lesion in the pleural cavity, particularly if it affected the visceral side.

I have been unable to find anything in the literature which deals with the venous blood supply of the bronchi in the presence of pleuropulmonary disease, nor is there any mention of the bronchoscopic aspect of this problem.

In his excellent monograph on the lung, Miller discusses the increase which exists in the blood supply of lungs which have a thickened pleura. In such instances, septa extend from the pleurae into the lung, carrying the blood vessels with them. Lungs with a thin pleura have no septa, though free venous circulation of blood between the bronchi and the pleura must be interfered with to account for the venous stasis in the bronchi in cases such as are being considered.

That tumefaction of the mucous membrane of the bronchus does not occur more often may be explained by the nature of the disease and the vascular arrangement of the individual lung. In all of these cases the congested mucosa bleeds when touched with the lip of the bronchoscope.

Two additional cases are cited to illustrate the value of this observation. A man, 51 years of age, was seen six weeks after his recovery from pneumonia, caused by the pneumococcus type 18. A few days before, he had coughed up about ten ounces of pus. His temperature was 101 degrees, his pulse was rapid and irregular, and he suffered with an incessant and distressing cough. X-ray study revealed an opaque shadow in the left lower lung field with a fluid level when taken in the lateral recumbent position. Bronchoscopy was requested in order to determine whether a bronchopulmonary condition existed. Bronchoscopy revealed a marked degree of passive congestion of both branches of the left lower lobe bronchus. As a result, the clinicians were told that a pleural cavity lesion existed.

After a preliminary thoracentesis, Dr. Lilienthal removed a large quantity of thick greenish pus mixed with fibrin from the pleural cavity. The patient was subsequently discharged in good health.

A man, 53 years of age, was referred by Dr. Le Fever, of Kingston, for bronchoscopy. Ten days before, the patient had suffered from what appeared to be an acute upper respiratory infection. In addition to having a fever, he coughed and experienced pain in the upper right chest during inspiration. Within 48 hours, his temperature returned to normal and he expressed a desire to return to work. His physician suggested that he submit to a fluoroscopic examination of his chest. The only item of significance in the history is the fact that during the past three or four months he had not felt like doing any more than necessary.

Bronchoscopy revealed a moderate degree of passive congestion in, and around, the orifice of the bronchus of the right upper lobe. Because of this, the attending physician was advised that the neoplasm which he suspected was in the pleural cavity and therefore inoperable.

A new bronchoscopic diagnostic sign for certain diseases of the pleural cavity is offered.

136 E. 64TH STREET.

CXII

CARCINOMA OF THE ESOPHAGUS: SOME OBSERVATIONS WITH TWO CASE REPORTS

WILLIAM A. HUDSON, M.D.

DETROIT

The revival of interest in the surgical treatment of carcinoma of the esophagus and the comparatively small number of cases that can be reported as being cured are, I think, sufficient reason for placing this report in the literature.

There is apparently considerable variation in the incidence of carcinoma of the esophagus in various sections of the world, yet it would appear that approximately 5 per cent of all malignant disease occurs in the esophagus.

Watson[1] has reported on the distribution of growths in the esophagus as they were encountered at the Memorial Hospital. He found 18.4 per cent occurred in the upper, or proximal, third of the esophagus. These figures are not at great variance with those of other authors. From these figures it is readily seen that carcinoma of the proximal one-third of the esophagus is a relatively frequent occurrence. It would seem desirable that any evidence of its curability should be recorded.

Past experiences with radiation therapy have been without encouragement. To a patient suffering with carcinoma of the esophagus, surgery has afforded the one hope of a cure. It falls to the lot of the surgeon, therefore, to do all in his power to establish earlier diagnosis of carcinoma of the esophagus and to institute surgical measures as early as possible.

Our report today deals with two cases of carcinoma of the proximal portion of the upper third of the esophagus. These cases illustrate in a dramatic manner the value of complete removal of the carcinomatous lesion and the disappointment that can follow anything short of complete removal.

The lesions involving the proximal portion of the upper third of the esophagus are so intimately associated with the larynx, or involve it in such a manner, that both the esophagus and the larynx must be removed if a cure is to be expected.

We make it a policy now to discuss our findings frankly with the patient and his relatives. They are told the diagnosis, the location of the lesion, and the seriousness of the outlook. They are then advised that, because of the involvement of the common partition, removal of the proximal third of the esophagus and the larynx is the procedure of choice.

REPORT OF A CASE

The first case is that of M. K., female, white, aged 57 years, who first came under observation March 30, 1939, complaining of sore throat and increasing difficulty in swallowing, with a moderate loss of weight. Direct laryngoscopy revealed the presence of an infiltrating mass involving the posterior surface of the posterior commissure of the larynx and extending over into the esophagus. A biopsy was taken. Microscopic sections revealed squamous cell carcinoma, not graded. The patient and her relatives were informed of the findings and were advised that a removal of the larynx and esophagus would be the procedure of choice if surgery was to be carried out; that anything short of this would probably fail to give a satisfactory result.

On April 13, a low tracheotomy was performed under novocain anesthesia. On April 22, under nitrous oxide and oxygen administered through the tracheotomy wound, double skin flaps were formed by making transverse incisions. The first was made at the level of the hyoid bone, and the second at the level of the lower border of the cricoid cartilage. A midline incision connected the two transverse incisions. Two lateral skin flaps were prepared, carrying with them muscle and all soft tissues down to the larynx. All blood vessels leading to the larynx were ligated by transfixion sutures and the larynx was removed in block.

It had been our hope that we might be able to leave a portion of the esophagus. Therefore, the esophagus was not removed with the larynx; but upon examining it, it was found that the carcinomatous growth involved a fairly extensive segment, and that it would be necessary to remove that portion of the esophagus lying above the third tracheal ring as well as the lower pharyngeal mucous membrane. This was done without incident and with a minimal amount of bleeding. The cut end of the esophagus was then drawn upward and attached to the right edge of the skin at the lower transverse incision. The right and left skin flaps were snugly attached to the prevertebral muscles so as to form a funnel leading from the pharynx down toward the open end of the esophagus.

The trachea was closed over, and a vaseline gauze pack placed over it as a protection against the leakage of secretion into the trachea. A duodenal tube was inserted into the esophagus for feeding purposes and the patient returned to her room. During the operation the pulse and respiration showed very little change. The pulse ranged between 80 F. and 100 F. throughout the procedure.

During the first week, the temperature ranged as high as 102 F., but there was no respiratory distress and no evidence of respiratory infection. The patient was kept in a position that placed the head lower than the body, thereby promoting drainage of secretion toward the mouth rather than toward the mediastinum. Her temperature gradually dropped, until on the sixteenth day it was approaching normal, with a pulse of 80 to 90, and a respiration of 20. Feedings were carried out through the duodenal tube and the patient was discharged from the hospital without complications.

It has now been three years since this operation was performed. The patient is able to swallow her saliva, but we have not completed our anterior wall of the funnel that was formed by skin flaps. There is some narrowing of the junction between the pharynx and this funnel which interferes with the swallowing of food. The patient is happy and contented. She maintains a good weight and is quite active.

REPORT OF A CASE

The second case is that of M. D. Y., white, female, aged 55 years, who complained of difficulty in swallowing, loss of weight, general malaise and chronic sinusitis. The onset of the difficulty in swallowing occurred in October, 1940, but about nine months later patient noted that her throat was constantly sore and that she had a hacking cough. By August there was considerable loss of strength and on admission to the hospital December 30, 1941, the weight had reduced from 165 to 139 pounds. X-ray studies revealed an inability to swallow barium without aspirating into the trachea and an irregular mass filling the uppermost portion of the esophagus. Esophagoscopy revealed a fungating mass occupying a position five inches from the teeth and extending for a distance of one and a quarter inches distalward in the esophagus. There was definite involvement of the common partition between the larynx and the esophagus. The pathological report on the biopsy was squamous cell carcinoma, grade 2, with secondary infection. There were 3,910,000 red cells, and 4,200 whites. The pulse was 110; temperature, 100 F.

The family was advised of the findings and of the very grave outlook for the future. It was recommended that resection of the upper third of the esophagus and the larynx be carried out. There was some reluctance on the part of the relatives and the patient regarding the resection of the larynx, and considerably against our better judgment, we agreed that if at all possible we would leave the larynx. Accordingly, on January 3, 1942, under inhalation anesthesia, supplemented by sodium pentathol, injected intravenously, a midline incision was made for a low tracheotomy. An intratracheal catheter was then inserted through the tracheotomy wound, and the inhalation anesthesia was administered through this catheter. An incision was then made along the medial border of the left sternocleidomastoid muscle from a point near the angle of the jaw, downward to a point near the suprasternal notch. By careful dissection the tissue planes were separated. The larynx was rotated to the right and forward, so as to expose the underlying esophagus. The esophagus was freed as was also the lower portion of the pharyngeal mucous membrane. No difficulty was encountered except at the common partition between the esophagus and the larynx. Here, it was found that the tissue showed considerable induration and we were doubtful if we had removed all of the carcinomatous growth, although all soft tissues were removed to such an extent that the thyroid cartilages were left bare. Inspection of the laryngeal mucous membrane presented no evidence of involvement. We, therefore, left the larynx behind with the reservation that we would probably find it desirable to remove it at a later date. The space previously occupied by the esophagus was thoroughly dusted with sulfanilamide crystals and then packed with gauze. There was no evidence of involvement of regional lymph glands.

The patient left the operating room with little evidence of shock. She had received 750 cc. of saline intravenously during the operation. She was kept in a position that drained all secretions away from the mediastinum; i.e., the foot of the bed was elevated and this position was maintained for ten days or more. The immediate convalescence was entirely uneventful, except that on the fourth day she had a temperature of 103 F. which immediately disappeared following the expulsion of a heavy mucous plug from the right stem bronchus. She was discharged from the hospital able to swallow her saliva. She received her feedings through a duodenal tube inserted into the stomach by way of the distal segment of the esophagus which had been anchored at the lower edge of her wound.

In April the patient complained of a sore throat, and on direct laryngoscopy it was found that there was a mass of granulation-like

tissue on the posterior wall of the larynx which proved to be carcinomatous when biopsy was studied. About this time the patient developed a pneumonic infection. During the convalescence from this infection the recurrent lesion became very extensive and we were forced to abandon all hope of resection of the larynx.

These cases present almost identical problems; that is, in both cases the common partition was involved to such an extent that removal of either the esophagus or larynx individually was not sufficient to remove the cancerous growth in its entirety. In the first case, an extensive but clean removal of the lesion resulted in a nice recovery, and the patient remained well more than three years afterwards. In the second case, with a somewhat less advanced lesion, failure to remove the larynx has resulted in failure to cure the patient.

It is evident that metastatic involvement of the regional lymph glands occurs late. This simplifies the surgical problem to a considerable extent. It is self-evident, however, that the earlier the diagnosis is made and the earlier the carcinomatous lesion is removed, the better the prospects of a complete elimination of the lesion.

We would like to make a plea that all patients who complain of distress of any sort on swallowing be studied carefully; that they have not only x-ray studies of the esophagus, but that careful endoscopic studies be carried out. If, at first, no explanation for the discomfort can be found, repetition of the studies may later reveal the real cause to be carcinoma. Then without further delay the lesion should be removed completely by means of surgery.

CONCLUSIONS

1. Carcinoma of the upper third of the esophagus can be cured (eliminated) by means of surgery.

2. The larynx and esophagus must both be removed when the common partition is involved.

3. Early diagnosis followed by surgical intervention at an early date is most desirable.

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Society Proceedings

SIXTY-FOURTH ANNUAL CONGRESS

OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

(CONTINUED FROM THE SEPTEMBER NUMBER)

The Sixty-fourth Annual Congress of the American Laryngological Association convened in the Marlborough-Blenheim Hotel, Atlantic City, N. J., at 10 A. M., May 25, 1942, Dr. Thomas A. Carmody presiding.

Chemotherapy in Nose and Throat Diseases

DANIEL S. CUNNING, M.D.

NEW YORK, N. Y.

The status of the sulfonamides is changing so rapidly that new and improved combinations are being produced and tried continuously.

It is now over seven years since the introduction of the sulfonamide group of drugs to the realm of medical and surgical practice.

In 1936, sulfanilamide was the first one of the sulfonamides to be employed as a therapeutic agent. It acted as a specific against hemolytic streptococci. Since its introduction, no less than eighteen of these drugs have been introduced.

In 1938, sulfathiazole was introduced to the profession, which worked well on streptococci, pneumococci, and staphylococci; in 1939, sulfapyridine which acted miraculously well on pneumococci, on which sulfanilamide had little effect.

In 1940, sulfadiazine seemed to act well on practically all organisms and proved itself to be the least toxic of all the sulfonamides. This is the drug of choice at the present time, and seems to be just as effective against all the organisms the preceding ones were supposedly specific against.

The sulfonamides have been a great boon in the treatment of all severe infections of the nose and throat, such as deep infection of the neck, suppurative sinusitis, erysipelas, cavernous sinus thrombosis, Ludwig's angina, laryngo-tracheal bronchitis, edema of the larynx and meningitis.

All physicians prescribing these drugs should be familiar with the toxic reactions that may follow their use, such as hematuria, renal colic, formation of calculi, and agranulocytosis.

Only 10 per cent of all patients receiving these drugs show any toxic manifestations, so in severe infections do not fear the toxicity too much, but give the drugs boldly and in large doses early in the infection.

DISCUSSION

DR. SAMUEL SALINGER: There are instances in which the reaction of the drug may cause some confusion in diagnosis. A patient with an external suppuration over the nose following a nasal fracture was given sulfathiazole and at the end of two days developed chills and a fever. Having had a similar experience following the administration of sulfathiazole in a case of meningitis, I stopped the drug, but the chills and fever persisted. They persisted for 24 hours and despite a negative blood culture I put this patient on sulfadiazine, and within 24 hours I obtained a satisfactory result.

In our hospital there have been three cases in which there were repeated chills and fever following the administration of sulfathiazole. These subsided when the drug was withdrawn.

DR. FRANK A. SPENCER: There are three things about sulfadiazine that I would like to emphasize: First, I believe that in small doses it gives very good results in relatively small doses. I have been using sulfadiazine for some time in many infections which promised not to be very severe or very alarming, and I was able to get much quicker results than I would without the drug. Second, I get a high concentration very early, as Dr. Cunning has mentioned. Third, it is not very toxic, so that I am using it routinely for a great many infections in which I would not have used any of the sulfonamides six months or a year or two ago.

DR. SAMUEL IGLAUER: Dr. Schmidt [Schmidt, L. H. et al: Studies on Sulphonamide-Resistant Organisms, Jour. Pharmac. & Exp. Therap. 74: (Feb.), 1942, No. 6] of Cincinnati has shown that sometimes these organisms become resistant to the drug. In other words, he takes an organism from a patient, and if the patient has been treated

with any of these drugs he has found that in a certain percentage of cases the organism will grow in vitro in the presence of the drugs; so that in cases in which one does not get a therapeutic effect, it may happen that the organism has become resistant to the drug.

He also thinks that once an organism becomes resistant, subsequent cultures from that same organism remain resistant, and he worries a little bit about spreading around the community a strain of streptococcus which is not susceptible to the drug.

DR. CLAUDE C. CODY: Dr. Cunning mentioned the local effect of the drug. Of course, it has a systemic bacteriostatic effect. It likewise has an antiseptic effect when used locally. When used locally there is an analgesic effect in addition to the bacteriostatic and antiseptic action.

It can be used locally in laryngectomy cases, and it will cut down primary infection considerably. In addition, it contributes greatly to the comfort of the patient, as is also the case after mastoidectomy.

Also, in the treatment of the more or less serious conditions, as in acute follicular tonsillitis, it cuts down the length of the disease and contributes greatly to the comfort of the patient.

As Dr. Imperatori has pointed out, it does not seem to have quite the same beneficial effect in the nose. The 5% sodium sulfathiazole solution does cause an irritation of the mucous membrane of the nose.

I have not been able to see that the sulfonamide drugs have any particular effect in acute maxillary sinusitis or in the common cold. But chronic maxillary sinusitis, especially in those cases with a bad odor, I have had clear up by simply irrigating with sulfathiazole solution. I am not prepared to make any explanation as to whether those were coincidental, but in these cases it is worth using the irrigation for several treatments.

I would like to ask Dr. Cunning if he has noticed, as we seem to have done, that after giving the patient one of the sulfonamides he becomes more sensitive to reactions when later given a blood transfusion.

We have also noticed that in typing patients for a blood transfusion we cannot depend upon the customary period of thirty minutes, but find that it is necessary to use an hour in order to see if there is any agglutination or lysis. Also, there is now ample evidence

that sulfonamides do not have any effect on immunity. They neither shorten nor lengthen it. In the severe cases it is well to continue the use of the sulfonamides until the immunity would naturally occur, because if one does not, one can expect recurrence of the disease before the natural immunity develops to protect the patient.

DR. GEORGE B. WOOD: How seriously irritating are the sodium salts of sulfathiazole to the antrum mucosa or the nasal mucosa? An article recently published in the Journal of the American Medical Association described experiments on animals which showed that the necrosing effect of sodium salts was very severe. This writer thought it was due to the alkalinity.

DR. BURT R. SHURLY: I have found that sulfadiazine is the greatest favorite of all the various sulfonamide drugs and I would like to ask if Dr. Cunning's experience coincides with the fact that large doses in the beginning are much more valuable than a small dose continued, and whether the symptoms will light up again unless the drug is used for a long period?

DR. D. S. CUNNING: In answer to the question relative to the effect of this drug on transfusion, I do not know about the typing. That is done in the laboratory, but I do know that practically all the meningitis patients are transfused several times while they are getting large doses of sulfonamide drugs, and if there were any discrepancy I would certainly hear about it from the laboratory.

Hereditary Hemorrhagic Telangiectasia

FREDERICK A. FIGI, M.D.

ROCHESTER, MINN.

The observations recorded in this communication are based on experience with twenty cases of hereditary hemorrhagic telangiectasia. Epistaxis was the presenting complaint in all of the cases and without exception this originated from telangiectases in the nasal mucous membrane. All of the patients presented similar lesions in other situations, particularly about the face, lips, tongue, neck, chest and finger tips; but only in a few instances did bleeding occur from these and this was not of serious moment. Only local measures were found to be of value in controlling the nasal hemorrhages. Electrocoagulation proved to be most effective in this regard. Snake venom was of little benefit in these cases.

DISCUSSION

DR. JOHN J. SHEA: I have such a case. The patient's mother has had the condition to my knowledge for thirty years and it is also present in a niece.

In the older books the condition is classified as Osler's disease. It is certainly a disease of the capillaries. As far as the blood is concerned, the clotting time can be normal and nothing which affects the thrombokinase element or calcium element seems to have any influence.

Alcohol is very bad for these patients. Every time my patient starts to drink the least bit consistently he has a series of nosebleeds.

Adrenalin, by its local action on the capillaries, controls the bleeding immediately, but only as long as the adrenalin action lasts. Radium gives the longest relief of any of the local applications, but its use is limited.

Of the coagulants, the ordinary electrical coagulation is satisfactory, but really the diathermy seems to be better than ordinary coagulation because it operates more deeply.

These patients apparently have a vitamin C deficiency. This is the only vitamin which enters the question and it is a good thing to keep up vitamin C therapy.

For the anemias the liver extracts seem to be better than either copper or iron, and I have had no success in the treatment of telangiectasia with snake venom. After all, it is truly a question of controlling bleeding from diseased capillaries.

DR. D. S. CUNNING: I had a case in which the diagnosis was a little doubtful. This was a hypertensive patient with about six lesions in the nose. I was able to control these lesions with a sclerosing solution. Have you used sclerosing solutions, Dr. Figi?

DR. FRANCIS L. WEILLE: Because this disease seems to be rare, I would like to tell you about a case that I happened to see.

The patient with this disease had been bleeding on and off for a number of weeks. After listening to this paper, my hair almost stood on end when I realized we had made the diagnosis by biopsy. The blood spurted from side to side. However, the diagnosis was made, the nose was full of crusts with a big hole in the septum and the patient bleeding, and what were we going to do about it?

After a great deal of effort we found that the bleeding was coming from the left lateral wall of the nose, so we decided to ligate the external carotid artery just below the ascending pharyngeal. The patient stopped bleeding for a while and then started again, and then we ligated the ethmoidal arteries. That stopped the bleeding and it had not recurred by the end of about three months, when I last heard of her.

DR. CHARLES IMPERATORI: In my whole career I recall four cases of hereditary telangiectasia, particularly of the nose. Of these four patients one died and the other three are still alive.

In this particular patient, after the destruction by diathermy, there occurs a regrowth in a different place within a month, and in fact, at times these little telangiectatic spots recur in the coagulated area.

The thing that has controlled this bleeding as much as anything is cotton impregnated with tannic and gallic acid. He inserts this into his nostrils loosely and within two or three minutes the bleeding ceases.

I did a submucous resection on every one of these patients and the operation stopped the bleeding because it was mostly on the septum, but now practically all the telangiectatic spots are on the turbinates.

There is a preparation known as "stryphnon" which I instructed two of these patients to blow into their noses or to aspirate into the nose. They take a paper straw and stick it into the stryphnon powder and then insert it into the nose and inhale.

DR. GORDON BERRY: May I ask whether there is any limit to the time of electrocoagulation which could be properly employed?

DR. ARTHUR PROETZ: I would like to ask Dr. Figi whether the location of new lesions has anything to do, in his opinion, with the procedure which is used to stop the old ones, that is, do they cluster about the old scars or would they occur where they do in the natural course of events?

DR. GEORGE B. WOOD: I have in my mind a patient, not with one of these hereditary telangiectasias, but one who had a bleeding spot on the septum in Kiesselbach's area, to control which I tried cautery, chromic acid and partial resections. After the elevation of

the mucous membrane on one side I did not take out the cartilage. This stopped the bleeding for seven days. The patient came into the office one day and said that was a fine operation I had done. I took the applicator and touched the spot and the blood spurted out. Without letting her see the blood, I put cotton in and cut above the lesion so as to cut the descending branch of the ethmoidal artery, and she never bled again. I had cut the nasal branch which comes down on the septum from the anterior ethmoid.

DR. F. A. FIGI: As Dr. Shea has stated, the disease was formerly referred to as Osler's disease. In fact, he was one of the first to describe it, though not the first. It had been previously described by a Frenchman by the name of Rendu [Rendu, M.: Bull. et mém. Soc. de Hôp. de Paris. 13:731-733 (Oct.), 1896], and not infrequently the disease is referred to as the Osler-Rendu syndrome. And in recent years, a man by the name of Goldstein has attached his name to the syndrome because he reported several cases.

We have used radium somewhat, but we have been trying to avoid it because it produces dryness, crusting and atrophy of the mucous membrane later; then, after some years, if the dosage has been high there is a secondary telangiectasia from the radium itself. This, in my opinion, has tended to increase the tendency to bleed rather than to decrease it.

What Dr. Shea said concerning the effect of alcohol in increasing the bleeding tendency certainly seems very logical since alcohol is a vasodilator, especially on the body surface. I was not aware of the deficiency of vitamin C in this connection.

I have not employed sclerosing solutions in this connection. I use them commonly in treating angiomas, especially those of the cavernous type. Recently I have been using them in capillary angiomas.

I would think one would experience considerable difficulty, however, in injecting the sclerosing solution at the site where it would be effective, for most of these sclerosing solutions should be injected intravascularly in order to be effective.

Submucous resection is commonly used as a therapeutic measure in this connection. The procedure sounds rather illogical but, as stated, these patients do not have a prolonged bleeding time. They stand general surgery quite as well as a perfectly normal patient. There is no difficulty with the coagulation of the blood. The defect is in the walls of the superficial capillaries and venules, many of

which consist of only a single layer of endothelial cells with a markedly thinned out mucosa stretched over it.

Biopsy should cause no particular difficulty, except that one might get pretty active bleeding, especially if he happened to strike the site of one of these lesions. I see no contraindication to it at all, especially since submucous resection can usually be carried out without any difficulty.

Concerning the time limit of electrocoagulation, I do not think there is any. I think it is largely a matter of the patient's tolerance. I have personally, on more than one occasion, electrically coagulated these lesions in from one and one-half to two hours, except for the periods that I had to stop to recocainize. The patient would bleed at new areas or would bleed around the periphery of the crust which the coagulation produced, and it was only by very intensive electrocoagulation that the bleeding could finally be stopped.

I feared that we would have some saddleback nasal deformities result in some of these cases. To date, I have not seen any.

I did not quite catch your question, Dr. Proetz, I am sorry.

DR. ARTHUR PROETZ: The question was whether the nature of the interference by blocking off the arteries might have any effect in starting new lesions or whether they always started independently in another place.

DR. FIGI: The lesions tend to become more numerous even though no treatment is carried out. This probably answers your question, and even though the lesions which are present are completely sclerosed with electrocoagulation or whatever agent one may use, these same new lesions which would occur otherwise do develop and they require the same treatment which has been necessary previously.

Some External Nasal Deformities and Methods Used in Their Repair

WARREN B. DAVIS, M.D.

PHILADELPHIA, PA.

Ill-advised inaccurate articles on plastic surgery in lay publications are deplored because of the tendency which they exert toward increasing self-consciousness in many hypersensitive individuals with insignificant deformities. Operations on such patients seldom in-

crease their happiness or decrease the introspective tendencies. Real nasal deformities, however, are numerous and their corrections are usually gratifying both to the patient and to the surgeon.

Slides of the following conditions were shown:

a. Congenital non-syphilitic deficiencies of the dorsum of nose, corrected by autogenous rib-cartilage transplants.

b. Lateral deviation of the nose from old fractures, corrected by partial submucous resection of septum, division of the fronto-nasal processes of maxillae, then placing the nose in the midline by digital pressure.

c. Excessive convexities of the dorsum nasi—usually associated with elongation of nasal tip.

d. Rhinophyma reduced to approximately the size of a normal nose by multiple elliptical excisions.

e. The loss of external nasal soft tissue repaired by pedicled flaps from the naso-labial folds. More extensive losses repaired by delayed frontal flaps, single or double epithelialized as may be required.

Accuracy in outlining flaps, gentleness in handling tissues and evenness in the approximation of subcutaneous tissues and skin margins, all tend to minimize scar tissue and thereby assist in securing the best functional and cosmetic results.

Laryngo-Tracheo-Bronchitis in Children: Classification and Differential Diagnosis

T. Roy Gittins, M.D.

Sioux City, Iowa

I. Infectious Types:

A. Non-specific: streptococcus, staphylococcus, influenza bacillus, pneumococcus, etc.

B. Specific: diphtheria.

II. Traumatic Types:

A. With demonstrable foreign body.

B. Without demonstrable foreign body.

1. Particles solid matter in mucus, too small to be identified (possibly peanut, popcorn, foods).

2. Liquids—oils (nose drops), medicines, milk, chemicals.

III. Allergic Types.

A. Edema (angioneurotic).

B. Asthma.

IV. Spasmodic Types (croup).

In this short paper, an effort is made to lay some stress on the importance of classification and differential diagnosis in the consideration of acute laryngo-tracheo-bronchitis, especially as this disease in its various manifestations afflicts infants and young children.

DISCUSSION

DR. FRANK R. SPENCER: Very few of the cases which I see are mild. Many are almost moribund when admitted. Certain features of these cases should be emphasized.

The tracheobronchial secretions are so dry and tenacious that it its impossible for the patient to expel them by coughing.

Last year, before the Nose and Throat Section of the American Medical Association, I emphasized the drying effect of some of the sulfonamide drugs. Their use adds to the difficulty these children have in breathing.

To make a bad matter worse, some are given morphine or codeine before being sent to the hospital. The warning of Dr. Chevalier Jackson not to use these sedatives has either not been heard of or goes unheeded.

A tracheotomy may not be necessary in the mild cases, but in the severe ones one often waits too long before the operation is performed. A tracheotomy of election is a conservative, life-saving operation and is not as radical a procedure as some general practitioners think. It should not be postponed until it becomes an emergency.

Forcing fluids and steam inhalations make tenacious secretions less adherent and expedite bronchoscopic aspiration. Most of these

patients are pretty badly dehydrated. They need the help of the pediatrician.

The laryngo-tracheo-bronchitis caused by a foreign body presents similar, but sometimes quite different problems. I had an unusual case last winter of a Mexican baby sent to the hospital for acute tracheobronchitis and laryngitis. This was due to a small cork screw which had been embedded in the upper end of the esophagus for six months. I believe any other child would have died long before, but this Mexican child, probably three-fourths Indian, had survived. The cork screw was removed and the child lived for a number of months. There was more and more obstruction of the esophagus until a gastrostomy for feeding had to be done, and about the time we attempted retrograde dilatation of the esophagus, the child died.

DR. C. L. JACKSON: I would like to ask Dr. Gittins whether he has any cases in which the children inhaled zinc stearate. How serious would that be; what has he done and how should that be handled specifically?

DR. T. E. WALSH: We have seen in the last month about seven cases of the infectious type with one fatality, and we feel that the good results were probably due to early tracheotomy. The child was not allowed to exhaust itself before something was done to help. We also attribute our good results to the administration of large amounts of parenteral fluids at once, and to the use of sulfadiazine. We feel that sulfadiazine is exceedingly useful in these cases. We have not found that the sulfonamides have caused much drying. However, we did find that oxygen should not be administered routinely in these cases because oxygen causes much more drying of the secretions. We have had to pull out thick plugs from the trachea and bronchi when we used oxygen. Lately we have omitted using oxygen entirely and give carbon dioxide. The patient is in a heavily steam-laden room.

DR. THOMAS C. GALLOWAY: I was much interested in Dr. Gittins' "traumatic" classification. I think it is something to which not much attention is paid, and I think it is important.

It is my own feeling that in an allergic condition we are much more likely to have a thick secretion, spasm, narrow lumen and edematous mucosa.

We should heed Dr. Walsh's remarks about oxygen, as well as Davison's [Davison, F. W.: Annals O. R. L. 49:1083 (Dec.), 1940]

fine demonstration of the value of mechanical humidification which produces saturated atmosphere at the temperature of 70 and not at a temperature of 85 and 90 previously used in the steam-room.

I hope Dr. Gittins will include under his "and so forth" postural drainage and irrigation, which I have written about, which I think is so important in the treatment of these cases, and which adds so much to the peace and the ease with which the bronchoscopist can deal with the condition.

In my service in the Evanston Hospital, which includes both private and charity cases, there have been nine consecutive recoveries without a death. That of course does not include the county hospital cases, which were not handled by myself personally in which the mortality has been much higher.

I think the use of postural irrigation and drainage in my hands has been the greatest source of comfort in treatment.

DR. BURT R. SHURLY: I would like to ask what is the objection to intubation in place of tracheotomy?

DR. T. ROY GITTINS: Most of the discussers embarrass me because my paper was about classification and differential diagnosis. If we went into all the treatments for this condition we could spend a great deal of time. Practically nothing new in the way of treatment has been recommended recently.

None of us, I think, is ready to say definitely what we are going to do with the sulfonamides. There are so many other issues. If the airway is mechanically blocked, one does not have time to use any of these drugs and many of the patients die even before one can do a tracheotomy.

When it comes to treatment, we have to consider the type of case, its severity and especially the age of the patient because the mortality varies inversely with the age. In children under a year the mortality is very high. In children under six months practically all of them die before they get to the hospital. In the literature, the mortality varies anywhere from 11 per cent to 75 per cent, and one man wrote me not long ago that his mortality was 100 per cent. I think it was pretty courageous for a man to write and admit that. When one considers the wide range of mortality, it must be clear that we must be talking about a lot of different things.

The main point brought up in the paper was the differential diagnosis. We have all been treating the subject as though all these

cases were of one type. This deserves a lot of discussion, for it is possible that a good many cases are being included in this group that are the milder types, traumatic, allergic and what not. If we get too excited about these mild cases, a lot of the patients are going to have tracheotomies that are not necessary.

When it comes to treatment, I would like to refer to what Dr. Shurly said in regard to intubation and tracheotomy. I think it has been pretty well accepted that tracheotomy is by far the safer procedure. If you have a competent resident on the job and he is not any farther than the end of the hall, then intubation might be all right; but putting in an intubation tube which might come out before you reach the corridor might make for a greater emergency than if you had not put it in.

One may catch the disease in the tracheal stages, in the larynx or in the bronchi and it may end up in the smaller divisions of the bronchial tree with blockage regardless of the organism.

I am not too sure of the organism and I do not think any of us are. One can get cultures of streptococcus or staphylococcus from any nose. How do we know which is predominating and virulent?

Dr. Jackson, I don't know about the zinc stearate.

Upper Respiratory Manifestations of Dermatologic Diseases

V. V. Wood, M.D.

St. Louis, Mo.

The chief purpose of this contribution is to stimulate more interest among laryngologists in the respiratory tract manifestations of dermatologic diseases. This group of pathological processes have never been allotted the space they deserve in the otolaryngological literature, although they are constantly appearing in cases under our care and observation. Their description and treatment is much more adequately covered in the dermatologic literature.

I shall list as many of the diseases having exhibitions pertaining to the subject as feasible: attempt to describe some of the more important ones as best I can, and show slides (some plain and most of them colored) that I judge to be illustrative of certain conditions.

The following dermatologic diseases are reported to exhibit mucosal lesions of the respiratory tract that should be of interest to the otolaryngologist: Lichen Planus, Lupus Erythematosus, Psoriasis, Acanthosis Nigricans, Keratosis Follicularis (Darier's Disease), Erythema Multiforme, Dermatitis Herpetiformis (Duhring's Disease), Pemphigus, Epidermolysis Bullosa, Scleroderma and Porokeratosis (Mibelli). A positive diagnosis of the mucosal lesions in these conditions may hinge upon an accurate recognition of the true character of the accompanying skin eruptions.

Certain Aliphatic Compounds as Nasal Vasoconstrictors

ARTHUR W. PROETZ, M.D.

ST. LOUIS, MO.

Vasoconstrictors in common use, from cocaine to amphetamine and some of the more recent compounds, are all benzene ring derivatives. Recently Shonle and Rohrmann have shown that certain aliphatic amines having the amino group attached to the second carbon atom exhibit pressor action. The present author has investigated the effect of these compounds upon the ciliated epithelium in laboratory animals and the clinical effect upon human noses.

The first, 2-amino heptane sulfate, is a potent vasoconstrictor, somewhat slower in its effect than ephedrine, but persisting over a longer period of time. The second is 2-amino 4-methyl hexane sulfate, the action of which is similar to the last but somewhat greater in degree.

Both of these substances have several times the potency of ephedrine. They have exhibited no systemic effects in any one of a series of several hundred patients. In concentrations of one to four per cent in isotonic solutions they have shown no deleterious effects upon the ciliary activity of the nasal mucous membrane when applied over periods of time far in excess of those encountered clinically.

DISCUSSION

DR. J. J. SHEA: We have been doing some of the clinical studies on the 2-aminoheptane sulfate.

We have used a 2 per cent solution on about 100 nasal mucous membranes, comparing it with the various office preparations which

we have used in the past. Briefly our clinical review shows that it outlasts any of the ordinary routine office vasoconstrictors. The thing which struck me most is that it is just as effective in acute vasomotor cases as it is in acute inflammatory cases.

DR. NORTON CANFIELD: I would like to ask Dr. Proetz if there is any analgesic effect? Also, is there any incompatibility?

DR. THOMAS C. GALLOWAY: Are there any secondary effects in the patients from the use of these materials?

DR. JAMES A. BABBITT: I noticed that Dr. Proetz referred to the use of 2 per cent ephedrine. There seems to be a great deal of opinion expressed that ½ per cent ephedrine in saline is desirable.

Do I understand from the paper that the use of a 2 per cent solution of ephedrine is preferable?

DR. BURT R. SHURLY: Can this material be sterilized?

DR. A. W. PROETZ: The drug has no analgesic effect. Its incompatibilities are very few, silver nitrate being probably the only one. It is quite compatible with anything else which we need to put in the nose.

The 2 per cent solution has a slightly corrosive effect on the atomizer, but with the ½ per cent solution it is negligible.

The solution can be sterilized by boiling and does not disintegrate.

There are certain individuals in whom it does not work as well as in others; in about every twentieth case one finds a person who reacts very slowly to it. Its average action is a little slower than ephedrine and other vasoconstrictors. This apparently is what prevents systemic reactions. It remains in the tissues for a long time.

It will be on the market as soon as it has been thoroughly tried out by the clinical investigators. It will be called "tuamine" and will be put out by Lilly.

I mentioned in my paper that I had observed no secondary effects, and in the short length of time that I have been working with the drugs (6 months) I have found no patients who have become tolerant to it. It has been quite satisfactory.

As to the choice between 2 per cent and one-half per cent solutions of ephedrine, Dr. Babbitt, I routinely prescribe 2 per cent solution made isotonic with sodium chloride for my patients to use at home. It is very much more effective than a one-half per cent solution. The specific difference I found was a quick recovery following the use of the 2 per cent solution and a long drawn out recovery with the 1 per cent solution. This is especially true in allergic noses with a great deal of secretion. There the 1 per cent is pretty quickly diluted to a concentration too weak to be effective. In the displacement treatment I use a ¼ or ½ per cent solution as suggested by Shambaugh, and find it adequate. It remains in the sinuses for 8 or 10 hours. A 2 per cent solution remaining for that length of time would constitute a considerable overdose.

DR. T. E. WALSH: Does putting in an odor spoil it?

DR. A. W. PROETZ: I do not know. The solutions we have used have very little odor.

DR. L. G. HUNNICUTT: Does the patient develop a tolerance to it very quickly as he does to ephedrine?

DR. PROETZ: He does not, at least not in the six months that I have observed it.

DR. THOMAS C. GALLOWAY: I think you might give a historical summary of how the drug was developed.

DR. PROETZ: I do not want to give you the impression that I developed this drug. My report is only on its clinical use.

The drug was discovered in the eighteen sixties. Last year Shonle and Rohrmann—this is all in the paper—found out that this drug was a pressor. Then Chen and one or two of his associates put it to pharmacological and toxicity tests, and my own contribution is restricted to its clinical use and laboratory tests on ciliated epithelium.

DR. GEORGE M. COATES: Is the material stable?

DR. PROETZ: Yes.

Ganglion Cell Neuroma of the Pharynx

(Case Report)

JOHN H. FOSTER, M.D.

HOUSTON, TEXAS

Miss R. M. S., aged 22, consulted me January 27, 1941, on account of a mass on the posterior wall of the throat on the left side. Examination revealed a smooth firm swelling occupying the whole left side of the posterior wall of the pharynx. Operating was advised with a tentative diagnosis of lipoma.

The tumor was enucleated easily. The main mass measured 5.5 x 4.5 x 2 cm. To this was attached a tail-like appendage 8 cm. long. The tumor was sectioned and the inner portion showed numerous ganglion cells scattered and in groups. Some areas had "almost the precise appearance of small sympathetic ganglia."

The case is reported for the reason that no case exactly like this appears in the literature.

DISCUSSION

DR. JOHN F. BARNHILL: What nerve was that supposed to be?

DR. J. F. FOSTER: There was no direct connection with any nerve that I know of.

DR. BARNHILL: Could you have a neuroma without it having a connection with a nerve?

DR. FOSTER: I suppose the nerve tissue there had some connection with the sympathetic nervous system because it is very close to the superior cervical sympathetic ganglion tissue.

Rhinoscleroma Involving the Upper and Lower Respiratory Tract

(Case Report)

F. W. Dixson, M.D.

Cleveland, Ohio

The patient, a white male, aged 55, born in Jugoslavia. His air passages became obstructed necessitating a tracheotomy in 1902 and again in 1904. There was a prolonged history of interference in breathing until the time he came under my care in 1934.

On admission his breathing was labored. He was cyanotic and a tracheotomy was done. The nose was filled with firm warty granulations. The trachea was narrowed and filled with a similar growth. These were removed and radiation applied. The tissue report read "Rhinoscleroma." The Klebsiella rhinoscleromatis was recovered in pure culture on blood agar.

In the past eight years, surgery followed by radiation has been employed in the nose twice and the trachea three times. The nose being more accessible seems to respond more satisfactorily to treatment than the trachea. A complete return to normal is not expected for permanent changes have taken place, but the progress of the disease seems controlled and the patient is comfortable.

DISCUSSION

Dr. Louis H. Clerf: I had the pleasure of seeing this case. I have also seen two others in which there was no nasal involvement. In this patient there was a definite nasal involvement.

The question of therapy has been of interest because one of these patients has extensive laryngeal involvement. Tracheotomy has been done and he received irradiation in small doses at frequent intervals with a certain amount of relief. If the therapy is discontinued, the lesion apparently flares up rather promptly. The other case is quiescent and is getting on very nicely. I am interested to know about the status of irradiation therapy in these cases.

Vincent's Infection With Unusual Symptoms

(Case Report)

K. M. Houser, M.D.

Philadelphia, Pa.

A 60-year-old woman was found suffering from chronic ulceration of the pharynx, loss of weight, secondary anemia and general debility. The previous year she had been hospitalized on three occasions. Improvement was partial and transient each time. There was some mucopurulent discharge in both middle meatuses and radiographs showed both maxillary sinuses cloudy. Smears from the tonsils showed many spirilla and fusiform bacilli but those from the ulcers showed none. When seen on the last occasion there was extensive ulceration of palate and pharynx and the uvula had sloughed away. Red blood cells, 3,500,000; hemoglobin, 63%.

Since Vincent's organisms could be obtained only from the tonsils these were removed under local anesthesia with great trepidation and only as a last resort.

Ten days later the ulcers were almost healed and in a month they were gone. They have not returned.

DISCUSSION

Dr. Thomas C. Galloway: What treatment did Dr. Houser use for this infection?

Dr. Henry M. Goodyear: I would like to mention a preparation which I do not believe has been used very often in the treatment of Vincent's angina and that is a 10 per cent copper sulfate solution.

I have seen cases of gingivitis which did not respond when treated by dentists for weeks clear up in a week or 10 days when the gingiva was swabbed once daily with it.

Dr. T. E. Carmody: I have been using copper sulfate for a number of years, 10 to 15 per cent in water, and then I give the patient a 25 per cent solution in glycerine, to use himself. It works almost perfectly. It is the best treatment for Vincent's angina that I have seen either around the gums or in the tonsils.

DR. K. M. HOUSER: About the treatment, I think all the standard treatments with the exception of copper sulfate were used. They included perborate, hydrogen peroxide, irrigation of the crypts of the tonsils themselves. Incidentally, these tonsils appeared perfectly normal until they were pressed upon. It was not the typical Vincent's tonsil at all.

As a matter of fact, as this is reported as unusual, of course I am not sure whether Vincent's organisms caused the condition or not. I am reasoning backwards, but the sores cleared up with the use of mercuric cyanide. We also used silver nitrate.

As far as the differential count goes, I cannot give you the figures, but I would say it was not in any way suggestive of infectious mononucleosis. There was some leukocytosis. This is kept up over a long period of time.

Abscess of the Parapharyngeal Space: Massive Hemorrhage, Recovery

(Case Report)

W. E. GROVE, M.D.

MILWAUKEE, WIS.

This case concerns a boy of 11 years af age, whose tonsils and adenoids had been previously removed and who developed consecutively a cold in the head, a stiff neck, swollen cervical glands, an otitis media and a swelling of the lateral pharyngeal wall posterior to the posterior pillar. An abscess in the left parapharyngeal space was opened externally at which time the internal jugular vein was ligated. Six days later a hemorrhage occurred which was controlled by a tight pack in the external wound. Two days thereafter a second alarming hemorrhage of from 300 to 400 cc. of blood occurred from the external wound. The bleeding vessel was identified as the ascending pharyngeal artery which was ligated. The bleeding ceased and the patient recovered after a stormy convalescence including a mastoid operation.

DISCUSSION

DR. HENRY ORTON: I had two cases in which there was massive hemorrhage following a parapharyngeal abscess, and it is not the most pleasant thing to have happen to see your patient just pass right out from a massive hemorrhage.

Amyloid Tumor of Larynx

(Case Report)

LOUIS H. CLERF, M.D.

PHILADELPHIA, PA.

A large isolated amyloid tumor which had been present for several years and produced progressive hoarseness and dyspnea was removed by thyrotomy from the larynx of a man, aged 48 years. The tumor involved the right ventricular band and vocal cord and measured 4 x 3 x 1 cm. A similar tumor now is developing on the left side of the larynx and removal is contemplated.

DISCUSSION

DR. ERNEST M. SEYDELL: I wish to report a similar case, a man of about 62 years of age, in whom the growth was subglottic. It was not removed. He had irradiation and lived for a number of years, dying from pneumonia.

DR. FREDERICK A. FIGI: Dr. Clerf's case is unusual in that the involvement at the time the patient came under his care was unilateral. More frequently in my experience the involvement has been bilateral, and commonly there has been a diffuse infiltration involving both vocal cords, often the ventricular bands as well, and in some instances extending pretty well down into the upper portion of the trachea.

In a gentleman 62 years of age, whom I took care of recently, there was marked obstruction from the tumor at the time he came under by observation. He gave a history of progressive hoarseness and respiratory difficulty of approximately three years' duration.

Examination showed a diffuse infiltration involving both sides of the larynx, chiefly in the subglottic region, where there was quite a sizable mass attached to the inferior aspect of each cord, there being only a narrow chink between these masses. The bronchoscopists were unable to insert even a small bronchoscope.

We had to do an emergency tracheotomy because of dyspnea during the course of the examination. About two weeks later I explored the tumor through a laryngofissure and found a mass measuring about 1.5 x 1 x 2.5 cm., along the mesial and inferior aspects

of both cords. This was excised, and below this, to the level of the fourth tracheal ring, there was a diffuse infiltration of the subglottic portion of the larynx and the tracheal wall. That portion of the tumor I removed with sharp curettes until an almost normal-sized lumen was established.

A skin graft was then applied to reline the larynx. I removed the soft rubber dilator about 12 days later. At this time the tracheotomy tube could be corked and within a few days following that we removed it. The man's voice was rapidly becoming normal and he was breathing without any difficulty.

DR. C. L. JACKSON: I should like to mention briefly a case in which, like Dr. Figi's case, the tumor was bilateral and rather diffuse.

The patient was a school teacher about 30 years of age, who had had a very serious impairment of her voice dating back for about two years before the time I first saw her.

The first diagnosis I made was phonation with the ventricular bands, and I made that without having any explanation for its development. But when I saw the patient the next time, about two years later, I decided to do a direct laryngoscopic examination, and take a little tissue for biopsy. There had not been very much change in the tissue but I have slides which made the diagnosis of amyloidosis of the larynx. This patient has gone on now for another three or four years without any change in her symptoms.

The growth seemed too diffuse to attempt to remove. She did not have dyspnea, so tracheotomy was not necessary.

I should like to know what Dr. Clerf would advise in the way of treatment in a patient like this, in whom the lesion was not localized, not obstructive, and where we certainly had no prospect of improving the voice by radical excision.

DR. L. H. CLERF: I am afraid I cannot answer Dr. Jackson's question.

I do not know why we get amyloid disease with isolated or more or less localized deposits. I suppose it is a matter of waiting until they get worse, and then doing what Dr. Figi did.

DR. FIGI: Why not irradiation?

DR. CLERF: I have had no experience with it.

DR. FIGI: I think that used to be our routine treatment, then excision and skin graft.

A Case of Osteitis Fibrosa Cystica

(Case Report)

JOHN C. KERNAN, M.D.

NEW YORK, N. Y.

This report concerns a young woman, 21 years of age, a Porto Rican, who had had a large firm swelling over the left eye for about a year. X-ray films showed it to be a cystic mass occupying both frontal sinuses, extending far back into the left frontal fossa and into the orbit on the left side. At operation it proved to be a cyst with thick fibrous and bony walls.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, Feb. 2, 1942

THE PRESIDENT, DR. JEROME STRAUSS, IN THE CHAIR

The Present Status of Nasal Medication

NOAH D. FABRICANT, M.D.

(Abstract)

In recent years the use of nose drops and nasal sprays for the relief of nasal congestion in acute head colds, acute sinusitis and allergic rhinitis has become exceedingly widespread. Despite the popularity of this form of medication, very few nasal preparations are prepared for public distribution with sufficient scientific forethought to produce the most effective product possible from the materials at hand. Many nose drops and sprays still contain an undesirable oily base. Others disregard the importance of ciliary activity in maintaining efficient streaming mucus in the nose. Again, a third group omits recognition of isotonicity and the desirability of an appropriate pH factor. These factors are discussed at greater length in the paper. The use of sulfonamide nasal sprays is questioned and indicated on the basis of extreme alkalinity; it is felt that such solutions are extremely caustic and may cause irreparable damage to the nasal and sinus mucous membrane. Finally it is only through a thorough understanding of the role of nasal physiology that the creation of an ideal nasal medication can be encouraged.

DISCUSSION

DR. E. E. SERLES: Probably no class of preparations manufactured today is so widely used for self-medication as are nose drops. Dr. Fabricant was rather pointed in some of the statements he made concerning rhinologic practice and it might be well to consider what we wish to accomplish with nasal medication. Are we content with the simple process of irrigation; do we strive to effect complete sterilization of tissues; are we concerned with restitution of tissues

damaged by infection; or do we want a combination of all three? If there is any definite criticism of medical science it is the tendency toward polypharmacy—trying to do too many things at once. Do we wish to interfere with normal secretions; do we wish to stimulate normal secretions; or are we likely to let nature take its course after we have done as much damage as possible? Do we desire medication through absorption into the cells, as a means of creating a defense mechanism in their own entity? These are the things that appreciation of the pH value cannot reach. Without wishing to detract from the splendid work of Dr. Fabricant, I would like to call attention to the fact that he has pointed the way to deeper investigation of the physiology and to the unsatisfactory endeavors made in the field of nasal medication.

The early medications were mostly volatile substances, applied by inhalation, which is a cumbersome method. The pharmacists and manufacturers, in good faith, believing these volatile substances were what were desired by physicians, turned to the best and cheapest solvents. They are not water soluble; they are soluble in alcohol, but that is extremely irritating to the mucous membrane. Oils were chosen as an economic factor in order that the medication might be applied direct to the area without setting up the teakettle and funnel.

With reference to alkalinity, again we are confronted with pharmaceutic necessity. Many substances are not stable except in an alkaline medium, and stability of at least two years' duration is essential under widely varying conditions—as far south as the Canal Zone and as far north as Alaska. Stability, therefore, frequently becomes of greater significance than therapeutic efficiency.

Dr. Fabricant referred to the saline content of the nasal secretions. In order to establish pH with any degree of certainty one must know the constancy of the nasal secretion. There are fixed laws for the constancy of such substances as ephedrine and these must be correlated with the solution content in order to maintain a pH on the acid side. There are other variables in the nasal secretions than that of salt, which might equally well influence the pH value, how greatly we cannot say because we are not sufficiently informed as to the constancy of the constituents of the secretions.

Another factor to be considered is whether the medium is of colloid character or crystalloid character. This makes a tremendous difference in the variances we may effect. Administration of any colloidal substance tends to "damp down" or buffer the action of the solution. Should we consider the question of a long period of

duration so far as the product is concerned, and longer sterilization of the field, water soluble compounds might be considered. Pectin might also be added to the solution. It establishes a completely aqueous base for substances soluble in water and includes a substance which increases the density of the solution and more nearly attains an osmotic ratio in normal tissue. If that ideal could be attained all mechanical irritants of drug application could be eliminated.

In the use of vasoconstrictors or vasodilators it would appear that we are striving for reduction of edema. We should be cautious in preparing a product which might become a matter of bargain counter procedure unless adequate controls are established with reference to these powerful drugs. It is entirely possible that as studies similar to that of Dr. Fabricant's are extended some of these problems may be solved.

Dr. Thomas C. Galloway: This excellent presentation is perhaps just a beginning. It is well to know what should not be done to the physiologic nose, but the nose subjected to acute infection is not a physiologic nose. It still remains to study more in detail what processes are essential to recovery from infection and how treatment may aid. Some of the processes we attack by drugs may be curative. Edema in inflammations may, by diluting toxins and increasing secretion, be conservative; we may do harm by too early interference. Profuse secretion early may, by washing out infection or bringing more antibodies, or otherwise, be conservative. We have discovered that atropine, which stops secretions, may actually be harmful.

Certain infections, notably adenitis, are helped by X-ray, which apparently accentuates the processes of inflammation. We may establish scientifically that some treatments that have an empiric reputation may actually be valuable in producing reaction of inflammation, as for instance the use of mild silver protein packs, the use of magnesium sulfate in glycerin, the old chlorine treatment, etc.

Dr. Noah D. Fabricant (closing): I would like to point out the swinging pendulum of treatment. It is only about fifteen years ago that the current trend of rhinologic conservatism came into being. Prior to the advances made in the study of nasal physiology most of our therapeutic knowledge was based on anatomy, pathology and surgical anatomy. There was at one time an era of promiscuous nasal surgery. We younger men, however, were taught to be more conservative in establishing indications for intranasal surgery. On the other hand, I believe we may now be reaching

another stage, that of over-conservatism; this because of the present concept of nasal physiology. Recently, at the Academy meeting, one rhinologist just dismissed all forms of intranasal medication; he felt that x-ray and heat are sufficient treatment. In other words, he epitomizes the trend toward too much conservatism.

There are those who are convinced that most of us are doing a great deal of damage by putting anything in the nose. This has not been proved. On the other hand, perhaps we are effectively restoring the normal physiologic process.

Origin and Development of Pharyngeal Lymphoid Structure

A. A. ZIMMERMANN

(Abstract)

The mode of development of the pharyngeal lymphoid structures in the opossum was studied. Didelphys virginiana is phylogenetically an ancient and primitive marsupial. Well-developed palatine and pharyngeal tonsils are present, whereas lingual tonsils do not exist. The palatine and pharyngeal tonsils may therefore be considered as phylogenetically old lymphoid tissue.

The findings of this study were demonstrated by a series of lantern slides, and summarized as follows: The second pharyngeal pouches disappear during the first half of the twelfth day of intrauterine life. (Opossum embryos are born at the end of the thirteenth day of gestation, whereupon they gain entrance into the marsupial pouch, remain fixed on a nipple for the ensuing seven weeks and are known, during this developmental period, as pouch-young.) The origin of the palatine tonsils, therefore, is not related in any way to the fate of the second pouches, neither with their dorsal nor ventral portions. During the second half of the twelfth day the tonsillar sinus arises at the approximate site of the previously present second pharyngeal pouches. Its origin is due not so much to active epithelial proliferation and outpouching as to a pressure effect of two adjacent mesenchymal structures: (1) a mound-like mesenchymal elevation medial to the epithelial furrow of the sinus; this elevation constitutes the anlage of the tonsillar tuber; (2) a mesenchymal ridge which is developing lateral to the sinus and ventromedial to the base of the first pharyngeal pouches. This

mesenchymal fold or ridge is caudad to the lateral palatine process of the maxilla. It contributes to the formation of the soft palate which, in the opossum and in all marsupials, continues into the extensive palatopharyngeal plicae. The mesenchymal ridge might be designated as the "posterior palatine process."

The appearance of the anlage of the tonsillar sinus in the general region of the obliterated second pouches is incidental. In the newborn opossum (10 mm.) there is as yet no indication of lymphoid tissue around the epithelial sinus. Solid epithelial outgrowths from the sinus as earliest primordia or secondary tonsillar crypts occur in 14 mm. pouch-young. They acquire lumina after more than 30 mm. CR length is attained. The earliest differentiation of lymphoid tissue in the mesenchymal stroma surrounding the tonsillar sinus begins at about the same time (approximately three weeks after birth). Accumulation of considerable diffuse lymphoid tissue is noted in specimens of 43 mm. CR length, approximately 40 days old. The first primary lymph nodules appear at 60 days, which is near the weaning period. Secondary nodules or germinal centers are present in a definitely weaned specimen measuring 21 cm. All indications point to the differentiation in situ of lymphocytes from the mesenchymal stroma.

Infiltration of lymphocytes into the squamous stratified epithelium lining the tonsillar crypts is very marked in pouch-young 60 days old; some earlier indications may be observed in slightly younger specimens. The age corresponds with the weaning period. It is possible that the infiltration of lymphocytes into the lining epithelium of crypts, as well as the appearance of lymph nodules, is related to infection which must occur at about that time.

In the adult opossum the tonsillar tuber becomes incorporated in the lymphoid tissue of the medial side of the tonsil. The sinus is retained as a deep primary cleft from which arise 12 to 15 secondary crypts, closely surrounded by many lymph nodules. The tonsillar inlet faces the undersurface of the soft palate near the lateral pharyngeal wall. Macroscopically the palatine tonsils are easily recognized as two ovoid elevations in the faucial isthmus. They are about 7 mm. long and 2.5 mm. wide. Their greatest height is about 5 mm. There is no tonsillar fossa, as no secondary subdivision of the tonsillar sinus occurs, and there is no well-defined palatoglossal arch in the opossum. The terminal portions of the crypts consist of degenerated epithelium without lumen. Completely detached terminal crypts and subsequent regeneration were not observed. The pericapsular lymphatic plexus is richly developed. It is difficult to

ascertain whether any of the lymph channels are afferent to the palatine tonsils, as valves are rare in this plexus. The tonsillar capsule is very delicate.

The origin of the well-developed pharyngeal tonsil in the opossum is not related in any way to the presence of a pharyngeal bursa. A gland-like diverticulum of the anterior tip of the embryonic pharynx exists as a transitory structure during the latter part of the tenth day of intrauterine life. This embryonic pharyngeal bursa disappears completely long before any lymphoid tissue differentiates in the roof of the posterior nasopharynx. This process occurs in pouch-young approximately 20 days old, slightly cephalad of the ostia of the auditory tubes. The pharyngeal tonsil rapidly extends both craniad and caudad. In pouch-young at 60 days the pharyngeal tonsil constitutes an elongated lymphoid structure extending from the highest point of the pharyngeal constrictor muscle posteriorly to a level well rostral to the hypophysis. At that age the pharyngeal tonsil has a total cephalocaudal dimension of 3.4 mm.

DISCUSSION

DR. L. B. AREY: The branchial region of the embryo is a difficult region to study. A series of entodermal pouches is located here that started originally for the purpose of serving the vertebrate as respiratory organs. Some have been abandoned; others have been salvaged and turned to different uses. This series of derivative organs is sometimes referred to as a series of ductless glands. They make a strange list—the eustachian tubes, the tonsils, the parathyroids, the thymus, etc. The relation of the tonsil in this seriation has been anomalous. Dr. Zimmerman has indicated that there is serious doubt as to whether it fits into the series, and Levin has shown that in the pig the palatine tonsil does not lie laterally in the pharynx but on the soft palate. This is an illustration of tonsils developing in a region other than where the second pouch actually was located; hence, the tonsil of the pig cannot be a derivative of the second pouch.

The necessity of a beautifully graded series such as that of Dr. Zimmermann cannot be overemphasized. It can be seen that the second pouch is fading out in the first half of the twelfth day and the definitive tonsil is coming in in the second half of the same day. Only a perfectly graded series can show such a relation.

If the second pouch is not the direct antecedent of the tonsil, is the situation of the tonsil just a chance location? The pig's tonsil

lies in the soft palate region, but in addition, there are smaller tonsillar masses which might be the true tonsils. If the tonsil does not derive directly from the second pouch, it at least arises in the same location and, in some degree, from the same tissue. It might be a fine question to argue that there is first a pocket, that this smoothes out, and that in this same region a tonsil then grows. Admitting that the second pouch does disappear, why does the faucial tonsil appear in approximately the same site Kingsbury explained this as the result of its occupying a neutral location in a region otherwise subject to growth shiftings.

The speed of development in the opossum is one of the most dramatic happenings in vertebrate development. It resembles a chemical precipitation. In man, it has been found that the crypts in the human tonsil first appear at about the middle of the third month.

The literature states that there are 12 to 20 crypts connecting with the surface of the tonsil, but if reconstruction is carefully carried out, it will be found that at as early as seven months there are some 40 primary crypts present. This number remains constant until the tonsils begin to decline in old age. In the study made, a number of cysts and solid precysts were found, which were not seen in the sections of the opossum.

In the human tonsil, at the time of birth crypts have formed up to the fifth order, and some are very complicated. Just after birth there is tremendous atrophy, the ends break off, undergo cystic degeneration and ultimately disappear; the condition after birth is therefore much simplified. In infancy they grow out again and, in children, the complicated, branched formation is restored, which holds until old age sets in.

In the study of the human tonsil the expansiveness of the crypt system was calculated, and it was found that the surface area of the epithelium of all the crypts of a single tonsil was about 46 square inches, or a total of 91 square inches for the two tonsils. The total exposed area of the pharynx, if the epithelium is stripped off after death, is found to be about 7 square inches, which means that there is about thirteen times as much surface area in the buried epithelium of the two tonsils as in the whole surface area of the pharynx.

Some authors have remarked how fortunate it is that a series of mucous glands exists which flush out the crypts and keep them clean. That is not true, however, as only in rare cases do mucous glands open into crypts. The glands appear before the tonsils and

only rarely communicate with them. It is also said that there are no crypts in the pharyngeal tonsil, and that what is seen in cross-section is merely a series of folds. The folds, of course, do occur, but coming off from them are many funnel-shaped tubes connecting with mucous glands, which are seemingly too large to be ducts. If one admits there are true conical crypts in the lingual tonsil, then these funnels in the pharyngeal tonsil are essentially the same. Hence I would be inclined to say the pharyngeal tonsil does have simple crypts. They are extremely numerous, and the glands enter their apices in the same way as they do in the simple crypts of the lingual tonsil.

DR. A. A. ZIMMERMANN (closing): In my opinion the palatine tonsil develops at the approximate site of the previously present second pharyngeal pouch, and its origin is due to a pinching effect of adjacent mesenchymal structures—the tonsillar tuber and the posterior palatine fold which is concerned in the formation of the posterior portion of the soft palate. The general correspondence of this site with that of the obliterated second pharyngeal pouches is incidental. The origin of neither the palatine nor the pharyngeal tonsils has anything to do with previously existing pouches of the embryonic primitive pharynx.

In the opossum there are usually only about a dozen or fifteen secondary crypts. Unquestionably the palatine tonsil of the opossum, while highly differentiated, is a less complex structure than that of man. I found no secondary detachments of tonsillar crypts.

The palatine tonsil of the pig is not a homologous structure to what we call the palatine tonsils of other mammalian species. In the latter forms the so-called "palatine tonsils" really lie in the lateral wall of the pharynx (faucial isthmus), whereas in the pig they are truly palatine in position but lack a tonsillar sinus.

Otitic Brain Abscess

HANS BRUNNER, M.D.

(Abstract)

Five cases of otitic brain abscess observed within the past two years were described in detail. There were two deaths in the series. While the results might be considered as satisfactory, the essayist felt that faulty conclusions might be drawn. In reviewing 55 cases of brain abscess which he had observed during a period of twenty-two years, the results were different. Among these 55 cases there were seven traumatic abscesses of the frontal lobe and three metastatic abscesses; these, with four abscesses of otitic origin found at autopsy, must be excluded.

Included in the remaining 41 cases were 28 cases of temporal lobe abscess with 10 deaths (mortality rate 68 per cent); and 13 cerebellar abscesses with 11 deaths (mortality rate 84 per cent). It must therefore be admitted that the results in otitic brain abscess are not good.

Three reasons are advanced to explain the failures: first, in many cases diagnosis was made too late; second, many cases were seen for the first time when they were ante exitum; third, operation was usually performed immediately the diagnosis was made, without considering whether or not the abscess was in the latent stage. Since 1932 the rules emphasized by the neurosurgeon, to wait for the time of latency, has been observed. It has been found, however, that it is frequently difficult to recognize this period; furthermore that there are otitic infections of the brain which never pass into a latent stage. The fourth reason for failure was perhaps the faulty technic employed. Former procedures have now been replaced by standard neurosurgical methods. The approach to the abscess is always through an infected field unless the source of infection in the middle ear or the frontal sinus is entirely cured, spontaneously or by operation.

Experience to date does not permit one to say which neurosurgical technic is best in treatment of otorhinogenic brain abscesses. At present the open technic is employed in cerebral abscesses and the closed technic in cerebellar abscesses, and this will be continued unless through practical experience other methods of approach are found more desirable.

DISCUSSION

Dr. Adrien Verbrugghen: I believe there are criteria for deciding whether an abscess is encapsulated or not, the cell count in the spinal fluid and the presence of an advancing choked disc. I believe that if possible the abscess should be treated through a clean field. Any meddling, especially if there is no certainty of the abscess being present, is extremely dangerous, and in my opinion occasionally an abscess is produced by needling the brain through a contaminated field. No abscess may be found the first time, but three or four weeks later the symptoms are progressing and, when definite signs present, needling is again performed and an abscess is found which I think has probably been produced by the needling. I am firmly opposed to needling a clean brain through a dirty wound, unless one is positive about the diagnosis, and it should not be carried out without carefully walling off the dura. The greatest danger is of producing infection in the subarachnoid space.

Brain abscesses are getting very rare so far as I am concerned—possibly I have not been as successful as I should have been with them and, in this respect, I am somewhat encouraged by what I have heard tonight. I think their rarity is due to the sulfonamide drugs and to the reduction of osteomyelitis and of mastoid disease.

Dr. Francis Lederer: Otologists should try to meet neurosurgeons on some common ground on this question. To speak of operating through a "clean field" in a case in which one is not aware of the presence of a cholesteatoma, seems rather illogical. I have spoken to neurosurgeons who implied that when the abscess originated from a rather questionable source, the approach could be through the clean field, but in a case with a clear-cut ear history to proceed by way of the mastoid. It seems a more direct and logical route.

Dr. Brunner has presented some cases in the form of a confession of shortcomings. This is always helpful, and he deserves great credit for his sincere presentation. I believe, however, when he says that "this was our fault," that "we overlooked something," or regarded labyrinthitis as an hysterical manifestation, he is stretching it a little too far. Such cases as he has reported would be, I believe, just as confusing to a neurosurgeon. No one can say that the question will be solved by avoiding a mastoid focus, going by way of a clean field, so-called, and ignoring the original focus of infection. Cases with lateral sinus and jugular bulb thrombosis and labyrinthitis must be dealt with, and we cannot always wait until

we have 4 diopters of choked disc indicating a walling-off and the optimum time for surgical interference.

Perhaps we could produce better statistics if we waited for a walled-off abscess, but the cases Dr. Brunner reported did not come under that head. The statistics from other clinics may appear better, but they are dealing with ideal and select material. His cases came on in a stormy manner. To be sure we went along in a clumsy way in some instances, but there was nothing to direct one to the diagnosis of an abscess; there were no classical signs.

Furthermore, the mortality statistics given should not be misconstrued. They represent the experience of a lifetime, and many variations in treatment have been employed in the last twenty or more years in management. What we must realize is that in the five recent cases described, despite the difficulties, three patients recovered.

Dr. Hans Brunner (closing): I concur with the suggestion of postponing the operation on a brain abscess until it is encapsulated, but it is frequently very difficult to determine when this is the case. According to the literature, development of the capsule takes from one to six weeks, which is too variable to be of practical value. Further, so far as otorhinogenic abscesses are concerned, it is seldom possible to determine definitely the time of onset. Naturally, in traumatic abscesses conditions are more clear.

The choked disc as a criterion as to the stage of latency is in my experience very unreliable. This is proved by the five cases presented tonight. The patients in two cerebellar cases who died did not have choked discs, although one of them had one of the largest cerebellar abscesses I have ever seen. Of the two patients with cerebellar abscesses who recovered one had slight blurring of the margins of the disc on the affected side; the other had a normal disc. Only one case of the five had an actual choked disc.

Of greater value is the decrease of general symptoms—fever, leucocytosis of blood and spinal fluid, although here too the concept of latency is ill-defined so far as otorhinogenic abscesses are concerned. These abscesses may pursue a fulminating course without passing through a latent stage; also, the course may occasionally be very insidious, without production of conspicuous symptoms prior to the terminal stage.

Dr. Verbrugghen considers operation through an infected field dangerous, and believes that puncture of the brain through an in-

fected field may produce an artificial abscess. The former statement is definitely refuted by our cases. Some of these were certainly complicated, yet it was possible to cure three out of five, including two with cerebellar abscesses. Furthermore, at autopsy of the two who died, it was found that operation through the infected field did not produce a diffuse meningitis. I cannot agree with his second statement. Every otologist with surgical experience has seen ruptures of the dura, and has punctured the brain without hitting the abscess, and in the vast majority of cases neither a meningitis nor an artificial abscess ensued, provided proper care was given. There are reports in the literature of cases in which puncture of the brain produced not only hemorrhage but an actual abscess. However, these cases are rare, almost as rare as cases of osteomyelitis of the skull following drainage of an abscess through a sterile field. If one states the contrary, it is necessary to prove it by exact clinical and pathologic facts.

My statistics over a period of twenty-two years are not good, and I gave the reasons therefor, the chief of which was the inadequate surgical technic employed by otologists in the past. I admit that all the improvements in technic have been introduced by neurosurgeons and not by otologists, and it was the purpose of my paper to show that a proper combination of neurosurgical and otological methods will probably yield the best results.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of March 2, 1942

THE PRESIDENT, DR. JEROME STRAUSS, IN THE CHAIR

X-ray Studies in Mastoid Pathology

JOHN H. GILMORE, M.D.

(Abstract)

Definite information should be derived from x-ray study of the mastoid process: (1) anatomic information including the extent of mastoid development, the type and distribution of cells, the position of the sinus wall and the emissary vein, any unusual anatomic variations; (2) pathologic information including the presence of inflammation, recent, old, or both, bone destruction, if present, and where it is located, the presence of cholesteatoma or other neoplasms; (3) postoperative information including the extent of cell extirpation, the presence of persistent infections and granulations.

Since the advent of sulfonamide therapy, these studies are increasingly important. The hospital roentgenologist no longer sees acute fulminating mastoiditis, but peculiar chronic mastoid complaints and middle ear infections which have been resistant to treatment. Caution must be used in the correlation of clinical and x-ray findings when treatment with sulfonamides is being used. Serial study is of great assistance.

A third position is used in all mastoid examinations. In cases with a history of an acute or recent infection, the Taylor, or submentovertical, position has been used; in chronic cases a modification of the Chamberlain-Towne, or suboccipito-vertical, position has been used. From the latter much information may be obtained concerning the presence of cholesteatoma. A number of pictures were shown to demonstrate that disease in the mastoid may be overlooked if the third position is omitted.

Differential diagnosis of findings observed on the suboccipito-vertical projection has included:

(1) A large antral space, which from an x-ray standpoint satisfies diagnostic evidence of cholesteatoma but at operation renders none. Such cases have been believed to be cholesteatoma which had been spontaneously evacuated into the middle ear and auditory canal through a large perforation.

(2) Anatomically large antra. These are more apt to be present in poorly or underdeveloped mastoids.

(3) Chronic infection in and about the antrum without the presence of a cholesteatoma which may produce in time a large antrum. However, the margins will be indistinct and irregular.

(4) An emissary vein superimposed over the antral region. The vein may be so magnified as to give a false impression of a large antrum.

(5) Malignancy in the mastoid process which may give the appearance of an enlarged antrum. Surrounding destruction of an irregular nature and other clinical findings aid in the differentiation.

The essayist feels that x-ray studies of the mastoid process in obscure disease often identify localized bone destruction, may be the deciding factor for surgical intervention, may assist in detecting with a greater degree of certainty the presence of a cholesteatoma by the use of a third position in routine mastoid study, and are therefore an almost indispensable factor in proper management of a patient with a suspected acute mastoiditis who is under sulfonamide treatment.

DISCUSSION

DR. C. H. CHRISTOPH: Dr. Gilmore has taken up the subject of mastoiditis from the radiologist's point of view; I shall discuss it from the clinical standpoint. It is true that we can make a diagnosis of mastoiditis without an x-ray examination. The typical case of mastoiditis, with the protruding ear, the sinking of the posterosuperior bony wall, pain and tenderness, the elevation of the temperature, leukocytosis, etc., can be diagnosed by any medical student. It is the unusual cases which require x-ray corroboration—those with normal ear drums, normal blood count, no pain and little fever. Even after the diagnosis is made, it is essential to know the distribution of the cells, the development of the mastoid, the location and the extent of the area of necrosis, the position of the lateral sinus and the condition of the petrous pyramid.

At the Illinois Eye and Ear Infirmary during the past five years we have had more than 200 mastoid operations; of these about 30

per cent were radical operations. About 70 per cent of these were diagnosed by Dr. Gilmore as cholesteatoma, and with very few exceptions we proved his diagnosis at the operating table. I believe many such cases were missed prior to his use of the third position.

Another type which might be mentioned is the case of otitis media treated with sulfonamide. There is ofen an improvement in clinical symptoms, a lessening of pain and discharge, and a reduction of temperature, while the pathologic process is steadily progressing and goes on to bone necrosis. This Dr. Gilmore has proved by serial x-ray films. All the cases mentioned above have had from one to four x-ray films, and I think the serial studies are of the utmost importance.

Dr. Alfred Lewy: It was hard to convince me that the x-ray film is more reliable than clinical symptoms in determining whether or not surgery is indicated in these cases. So far as acute mastoiditis is concerned I am still unconvinced, except in patients who have had sulfonamide treatment. In many of these the indications are obscured, and when we have followed the interpretation of Dr. Gilmore we are glad we have done so. I would like to ask him if he has been able to confirm Law's observation that sulfanilamide causes any misleading appearance of the cell structure and may be misleading as to the actual status of the mastoid.

The one condition in which it has been particularly valuable is in the chronic suppurative cases in which we were in doubt as to whether surgery should be used or whether they should be treated conservatively. In the clinic we are inclined to treat these doubtful cases as surgical, because otherwise they would be neglected and something might happen when the patient was away from our care; these people do not come in regularly enough for treatment. Although a case can be treated successfully in the office by conservative methods, in the clinic one cannot always be sure of it.

Taking the base plate routinely was largely at my suggestion. This was done so that we might have the picture if the question of petrositis should arise. It has proved very valuable in the study of the antrum in the presence of suspected cholesteatoma.

Dr. John R. Lindsay: There is no question about the value of x-ray films in any type of mastoid suppuration. In the case of acute suppurative disease, it is well to realize what information we desire and expect to obtain from x-ray study. In the first place, we hope to determine the extent of the middle ear air cell system. In considering otitis media we should think in terms of a middle ear system

of air cells rather than simply a middle ear and a mastoid. We can expect the x-ray film to show the extent of the air cell system, whether it is limited to the mastoid, as occurs in two-thirds of the cases, or whether it extends into the perilabyrinthine or other regions of the pyramid. This is the primary requirement of a good x-ray examination of the temporal bone. Second, we hope to obtain information as to whether the air cell system is normal or diseased. Definite information as to whether or not cell breakdown has occurred is not always afforded with certainty by means of an x-ray film. However, it is only in rare cases that we depend on x-ray information to indicate the necessity for operation. The primary aim in an x-ray examination should be to demonstrate the air cell system in all its extensions.

DR. JOHN H. GILMORE (closing): Dr. Lewy asked about Law's observations of the trabecular intensification in the mastoid process following the use of sulfonamides in acute mastoiditis. We have observed this change. I started a series of confirming observations but just about that time we began to see very few acute mastoid cases. Relatively few acute mastoid cases are examined. Dr. Lewy mentioned the assistance we have had from the third film. We started to use the Taylor position but, as I could not see as much as I wanted of the chronically infected mastoid, I changed to the modification of the Chamberlain-Towne position.

I have seen petrositis in cases in which very little pathology was evident externally (no mastoid involvement). In such cases, information concerning the petrous bone may be obtained more readily by the Taylor position, rather than by the modification of the Chamberlain-Towne position.

I agree with Dr. Lindsay. In cases where the patient is clinically well immediately following acute mastoiditis, and where you cannot be sure that bone destruction is not progressing, x-ray examination is decidedly helpful.

Encephalomeningoceles in the Nasal Cavities

OLIVER B. MCGILLICUDDY, M.D.

LANSING, MICH.

(This article appeared in full in the June issue.)

DISCUSSION

DR. SAMUEL J. PEARLMAN: There is little to add to this nicely presented report. The total number of cases reported in the literature is few, and we should note that the essayist speaks only of meningoencephalocele, leaving out pure meningocele and encephalocele.

He is to be congratulated on being the third to report a living case and for his clinical acumen in suspecting what he had to deal with prior to operation. It seems to be true that the simple procedure of removing what appears to be an ordinary polyp may result in rapid death from meningitis. If no postmortem examination is held, the surgeon may blame himself unnecessarily. Hence from a diagnostic point of view, were every case of apparent nasal polyp disease submitted to x-ray examination, it might be possible on occasion to see a skull defect and so suspect an unusual condition.

It has been further suggested that under these circumstances, if air were injected into the suspect mass, none would appear in the ventricles in the case of an encephalomeningocele, thus differentiating it from a pure meningocele.

DR. HOWARD C. BALLENGER: Dr. McGillicuddy is to be congratulated on the successful outcome of this case. I understood him to say there is some difference of opinion as to the etiology of meningoencephalocele, that it might arise from an embryonic rest of the central nervous system. That could be true of an encephalocele or a glioma, without meninges, but it would seem that a meningoencephalocele would require a defect or a dehiscence in the base of the skull.

DR. HANS BRUNNER: Fortunately, up to the present time I have not seen a case of meningocele within the nose. However, I know of a case in which a meningocele was misinterpreted as an ordinary nasal polyp. Novocaine was injected into the mass and the patient died immediately.

So far as Dr. McGillicuddy's case is concerned, I should like to ask whether or not an encephalography was performed; also, I would

like to know how the patient reacted to an intravenous injection of hypertonic glucose solution. Both procedures are of interest as therapeutic factors: the encephalography by producing a slight irritation of the meninges, and the hypertonic glucose solution by decreasing the pressure of the cerebrospinal fluid. Finally, I would suggest that prior to further operation a trial should be given to deep radiation of the choroid plexus.

DR. OLIVER MCGILLICUDDY (closing): Dr. Pearlman was very kind in his discussion, because he has also reviewed the literature and I am sure could have pointed to many errors of omission in my paper. He was going to refer to a case of O'Brien's but did not, because it was not a true encephalomeningocele as it contained only meninges and cerebrospinal fluid.

Dr. Ballenger brings up the difference between encephalomeningocele and encephalocele. I should say that all congenital extracranial tumors containing brain tissue and meninges should be classified as encephalomeningoceles, whether or not they are attached to the brain by a pedicle. Cases have been reported which contained only brain tissue, and these should be classified as encephaloceles. If, as in O'Brien's case, there is only cerebrospinal fluid and meningeal tissue, the tumor should be called a meningocele.

Dr. Brunner brought up the question of encephalograms to determine whether or not the tumor was connected with a ventricle. This procedure would be of great value preoperatively. His suggestion of x-ray treatment to the choroid plexus is also an excellent one. Lempert suggested frequent spinal punctures to reduce the cerebrospinal fluid pressure. Prior to operation and for a short time afterward, this boy did not attend school because of the continuous drainage from his nose when he held his head forward. That has practically stopped, but as long as there is an opening I think he is safer with rhinorrhea.

As I stated, there must be many of these tumors that are missed. I have seen this case and one other that was similar. Eight years ago I saw a man in consultation who was dying of meningitis. At autopsy a large cribriform dehiscence was found. The general surgeon who had removed a large, apparently innocent polyp in his office was amazed at the opening through the cribriform plate. He had used some traction on the polyp but could not understand how he could have pulled away part of the cribriform bone. I think the obvious anwser is that he had removed an encephalocele, but we could not prove it because the polyp had been discarded without examination.

Histopathologic Changes of the Temporal Bone in Paget's Disease

M. TAMARI, M.D.

(Abstract)

The histologic structure of the temporal bone in Paget's disease is quite different from that in the other bones of the skull or those of the rest of the body. This is due to the formation of the labyrinthine capsule, which consists of three layers: the endosteal, built on a connective tissue base; the middle enchondreal; and the periosteal. In the early stages, the disease begins with a fibrosis of the marrow spaces; later there occurs destruction of the bone alternating with new bone formation, leading to the well-described mosaic structures.

Included in the study are three new cases of Paget's disease with bilateral involvement in each case. One case was complicated by an acute otitis media and a metastatic sarcoma.

The microscopic sections showed that the bony changes begin in areas best supplied with marrow tissue. The labyrinthine nucleus, because of its almost marrowless structure, is least susceptible to change. The periosteal layer was reconstructed in each case. The enchondreal middle layer resists for a time the rebuilding power. The greatest resistance was shown by the endosteal layer, the modiolus, the area cribrosa and the outer plate of the fallopian canal in its course through the middle ear.

The new bone approaches the labyrinthine inner layers in a humplike formation, which seems to be dependent upon the vascular supply. The resistance of the inner layer causes deformation of the spiral lamina, the inner surface of the cochlea, the vestibulum and the semicircular canal.

Paget's disease of the temporal bone gives the clinical picture of conductive deafness. It changes with progression of the disease to a mixed impairment and finally results in total deafness. At first the vestibular response is normal. Only in the later stages does it show a hypo-irritability of the balance apparatus. This is probably due to the fact that the changed bone conduction delays reaction to heat and cold.

DISCUSSION

DR. ELMER W. HAGENS: Dr. Tamari's complete paper gives an excellent detailed description of this unusual condition. I have no bones showing this but thought it might be interesting to compare

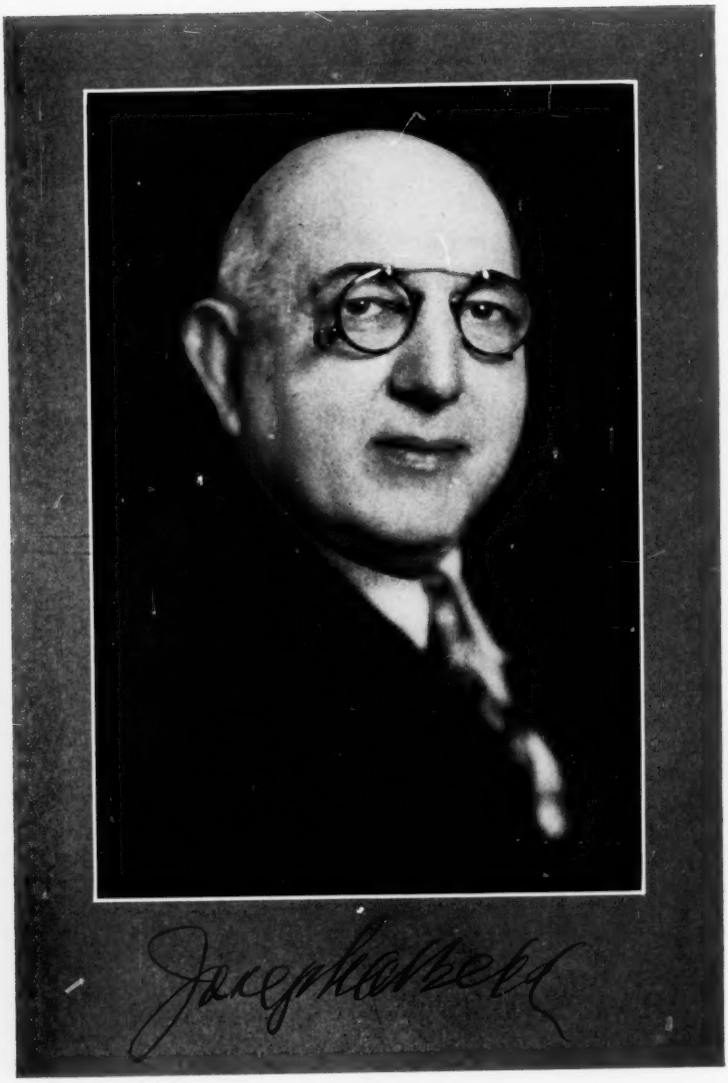
the condition with otosclerosis. A most important differentiation is that otosclerosis is a focal condition, while Paget's disease is diffuse. In some otosclerotic bones it will occur at the round window, the cochlea and the semicircular canal but between the foci you will find the old original bone. The early deafness of otosclerosis is through fixation of the footplate. When inner ear deafness occurs some other factor is working. To my knowledge no one has ever proven what causes inner ear deafness in otosclerosis. The factors Dr. Tamari mentioned as explaining deafness in his patients probably would not hold true in otosclerosis. He mentioned Haversian development in the new bone. In the specimens I have had this is very unusual. Bone cells are scattered, and where I did find Haversian systems it seemed they had been left over and were not a new formation. Cartilage remnants are hardly ever found. At the margin it is uncommon to find osteoclasts. They are found more in the focus itself, more centrally located. Otosclerosis progresses with finger-like projections from the focus. The marrow spaces are vascular and fibrous.

Paget's disease seems to occur more frequently in older men, whereas otosclerosis occurs earlier in adult life and more often in women. Neither disease seems to involve the ossicles; it has been rarely reported. I do not think otosclerosis such as we see in the mastoid is ever found there. Otosclerosis can grow to the periosteal layers or the endosteal layers, or both. The endosteal layer resists this process, and even infection for a long time.

Notice

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money is now available from the Casselberry Fund of the American Laryngological Association for a prize award, decoration or grant to be given for original investigation in the art and science of Laryngology or Rhinology. Theses must be in the hands of the Secretary, Dr. Arthur W. Proetz, 1010 Beaumont Medical Building, St. Louis, Missouri, before March 1, 1943.



JOSEPH CLAR BECK

1870-1942

Joseph Clar Beck was born September 26, 1870, in the small town of Dobrish, about 25 miles from Prague, in what was then Austrian Bohemia. He was the youngest of five children, the oldest a girl and the others boys.

They all eventually came to this country and settled in Chicago, where three of the brothers, Carl, Emil and Joseph, practiced medicine, and Rudolph practiced dentistry.

Previous to his arrival in America at the age of 14, the young brother, Joseph, had received very little education, yet when he entered medical school seven years later, he demonstrated an alertness of mind and a thirst for learning which soon placed him near the top of his class and which never deserted him throughout his long, busy career.

After his graduation from the College of Physicians and Surgeons, Chicago, in 1895, Doctor Beck assisted his oldest brother, Carl, in general surgery for three years. This association was valuable in many respects, but most of all it stimulated in the young physician an insatiable interest in pathology which remained a dominant factor in all of his medical activities. This interest took him in 1899 back to Czechoslovakia where he spent several months at the Pathological Institute, University of Prague. It was during his studies in microscopy of the temporal bone that he developed a fondness for otology and he immediately set about to prepare himself in the field of otolaryngology. This preparation covered a period of two years and took him to all the important medical centers of Europe.

Upon his return to Chicago, "Doctor Joe" set up an office with the other Doctors Beck, and this family relationship maintained up to the time of his death.

Doctor Beck's medical affiliations and honors were myriad and distinguished. He early joined the teaching staff of his Alma Mater, now known as the University of Illinois, College of Medicine, and served there as Associate Professor until 1932 when he was made Emeritus Professor. His teaching days were not over, however, and in 1940 he became Associate Dean of Medicine at the Illinois Eye and

Ear Infirmary, a position in which he was active until his recent illness.

Doctor Beck's other hospital appointments were: Attending Surgeon, Ear, Nose and Throat Department of Cook County Hospital for 18 years; Consultant at the Veterans' Hospital, Hines, Illinois, from 1927 to 1938, and during the past few years Senior Attending Staff of the Michael Reese, Henrotin, and Mount Sinai Hospitals in Chicago. He was one of the original examiners for the American Board of Otolaryngology and a member of most of the national and local societies which concerned his specialty and borderline fields. In 1915 he was made president of the American Academy of Ophthalmology and Otolaryngology, and in 1919-20, Chairman of the Section on Laryngology, Otolaryngology and Rhinology of the American Medical Association. He was president of the American Laryngological, Rhinological and Otological Society in 1933, and the American Broncho-Esophagological Association in 1936. Doctor Beck's membership in other special societies included the American Laryngological Association, the American Otological Society, and the Society of Plastic and Reconstructive Surgery. He was also a fellow of the American College of Surgeons and a member of the Chicago Laryngological and Otolaryngological Society, of which he was president in 1912.

During World War I he served as a Lieutenant Colonel in the French Division of the Czechoslovakian Army in charge of American Red Cross Hospital No. 113, at Cognac, France.

He was the author of a large number of articles and a few books, covering a wide range of medical subjects. Although most of these dealt with otolaryngology, many concerned borderline conditions, with particular emphasis on malignancies and reconstructive surgery.

Doctor Beck was a familiar figure at all national and local special society meetings. His presence was always felt at these gatherings and there were few indeed in which he did not take an active part. It has been said that no man in the specialty was more familiar with otolaryngologic literature than Joe Beck, and it has also been said that no one had a wider acquaintance with otolaryngologists the world over than he. There is evidence of this in his book, "Fifty Years in Medicine," in which he pays tribute to over 300 otolaryngologists from the various parts of the country, mostly intimate friends of his, most of whom called him "Joe."

Doctor Beck did much for otolaryngology but will best be remembered as an enthusiastic teacher and as a constant inspiration to

the younger members of the profession. His zeal along these lines prompted the organization of the five Joe Beck Reading Clubs in Chicago, which he regularly attended. It was at these meetings that his influence on the younger generation of otolaryngologists was most effective. Although he assumed the role of onlooker, he took an active part in the discussion of each subject and added color and zest to what otherwise often would have been a drab, uninteresting topic. He was the link between the past and present in otolaryngology, and the evening was commonly sprinkled with lovely anecdotes of other days.

His enthusiasm for these gatherings never dimmed, although his strength was ebbing and he was confined to his home. The last reading club meeting was held there in May with his leading the discussion as usual.

A final tribute was paid "Doctor Joe" by his "boys" who met as was their custom during the recent Academy meeting. A telephone hook-up was arranged so that he took part in the evening's activities although confined to his bed at the time. It was a fitting finale to a long, useful career. His old students heard his voice for the last time that night. Doctor Joe had attended his last meeting, his work was over, and he died a few days later.

O. E. V.